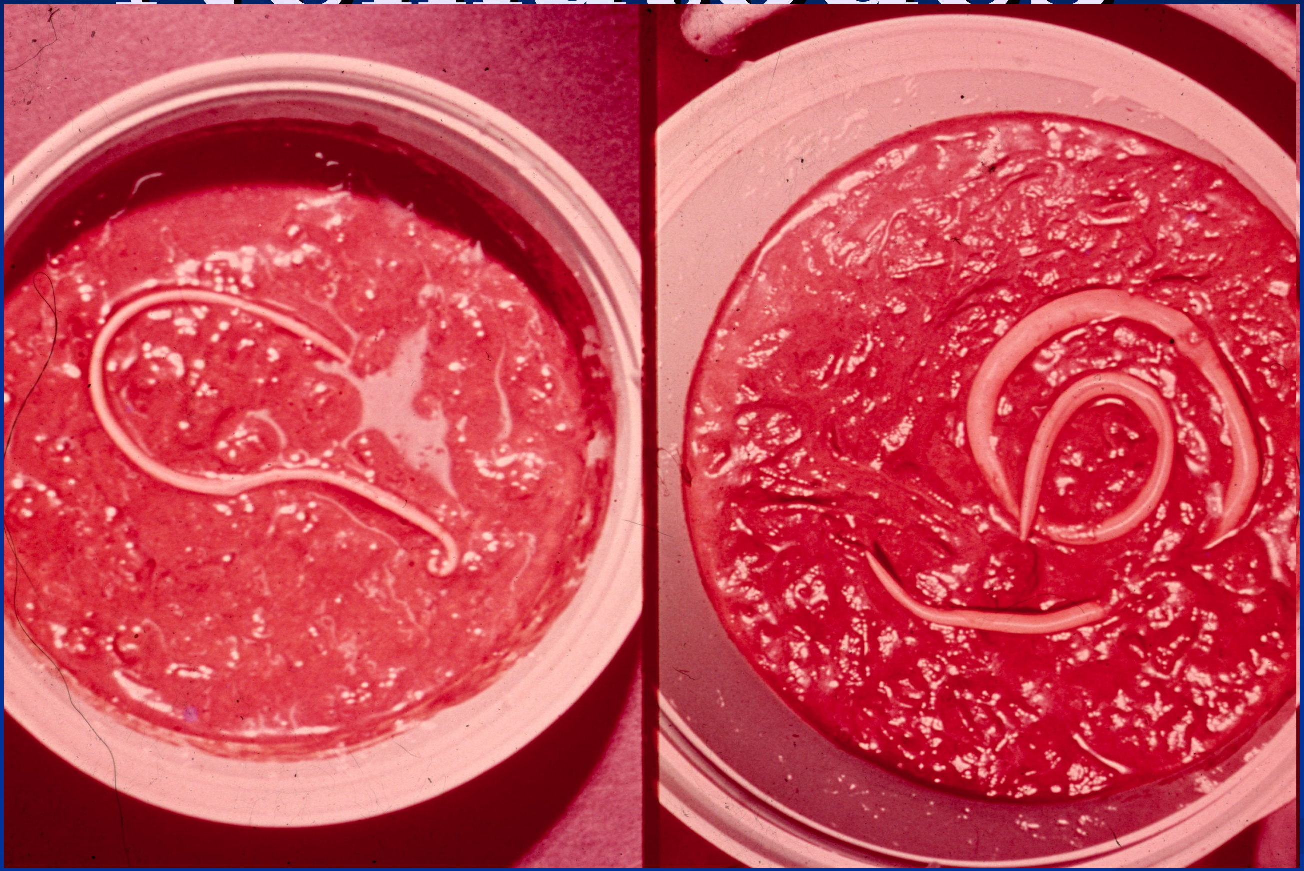
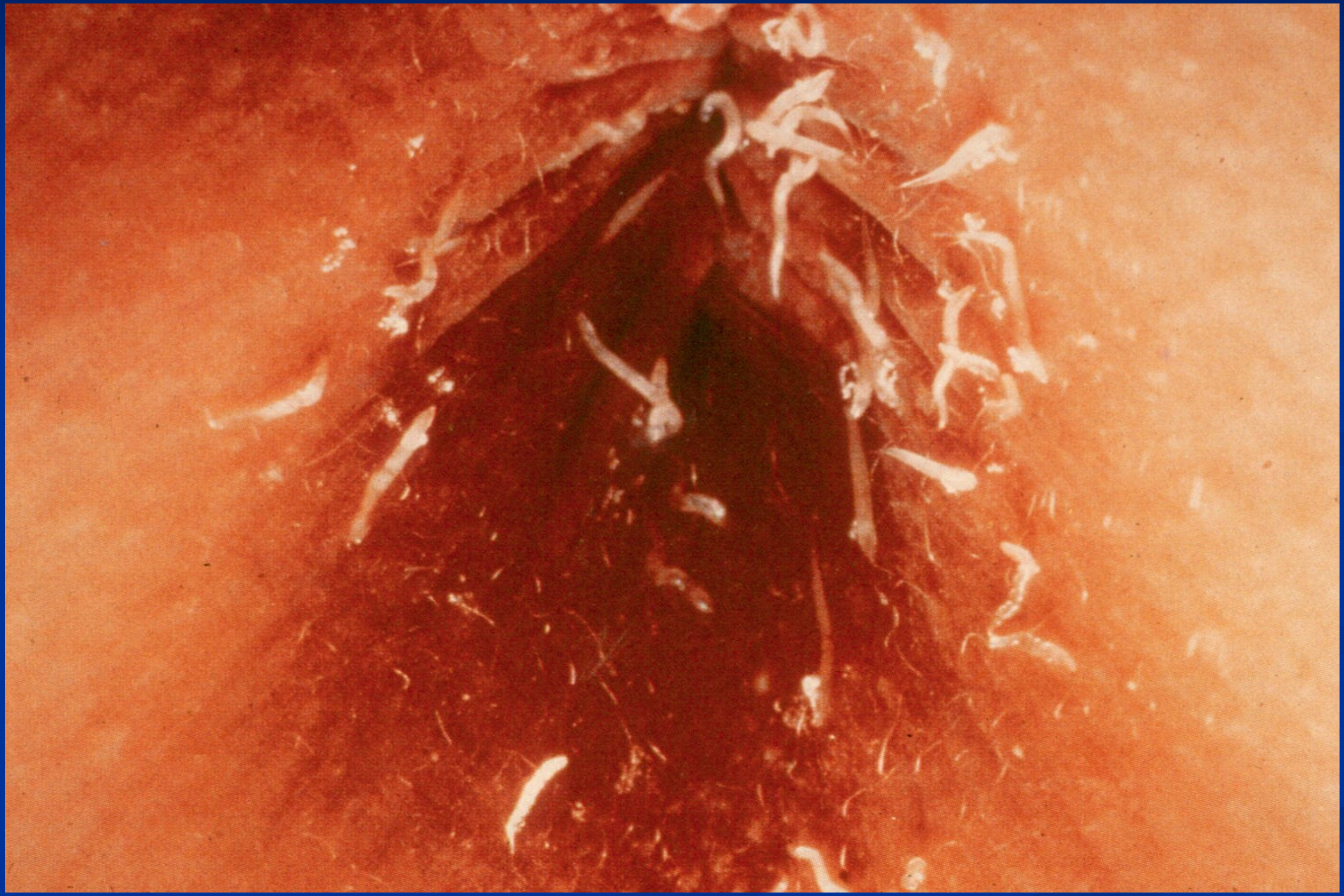
Nematodes

The Roundworms

Intestinal Nematodes



Pinworm



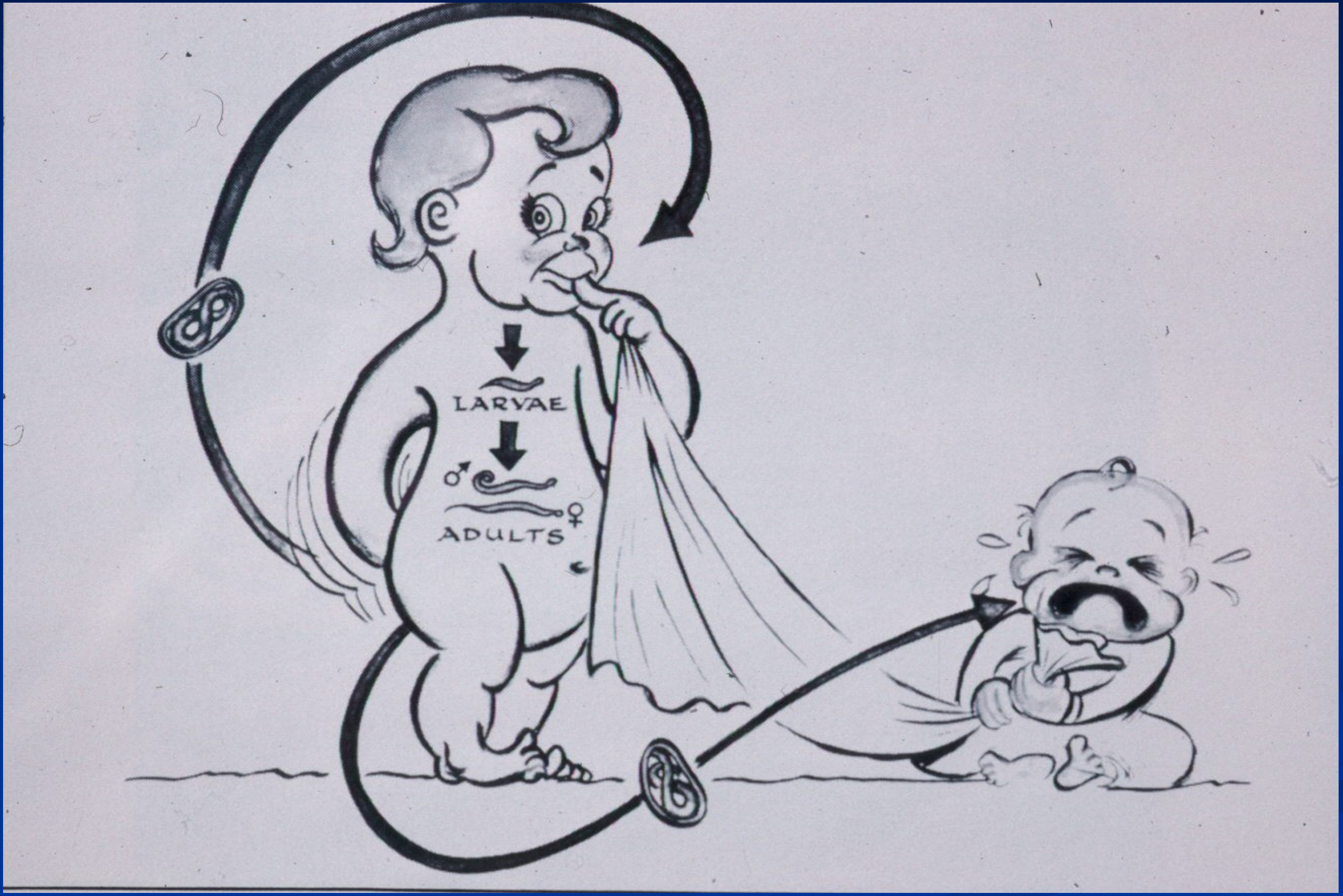
Pinworm Epidemiology

- *Enterobius vermicularis*
- Worldwide distribution, more common in temperate zones
- Most common helminthic infection in US
- Usually in children 5-10 yrs of age
- 20-30% of elementary school kids, 80% in crowded conditions

Pinworm Life Cycle

- Direct via ingestion of eggs from anus to mouth or from air, dust, sheets, clothing
- 1st stage larva hatches in duodenum, molts twice to 3rd stage on way to cecum, matures & mates
- Gravid female crawls to perianal area & lays 5-17k eggs
- PP 2 wks – 2 months

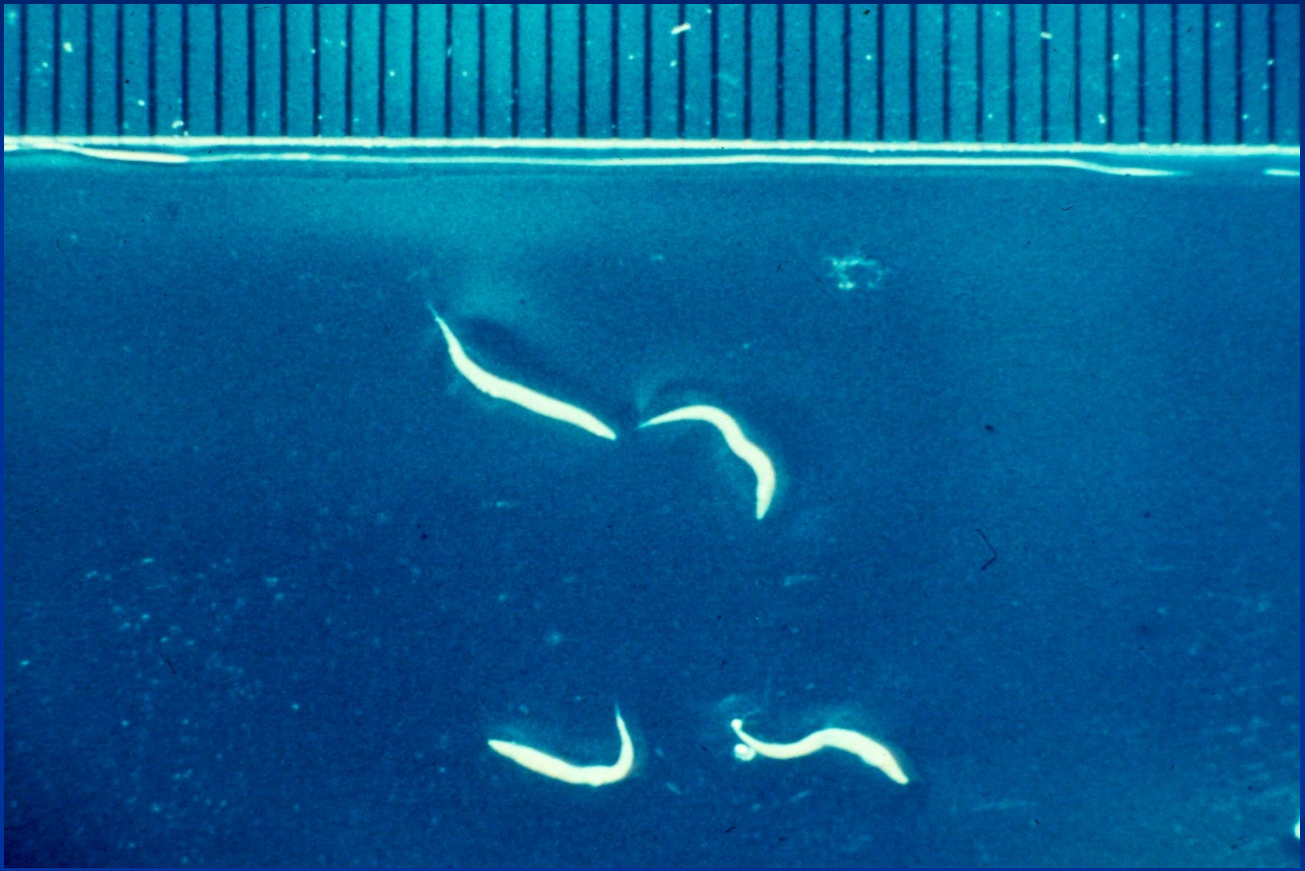
Pinworm life cycle


LARVAE
♂
ADULTS
♀

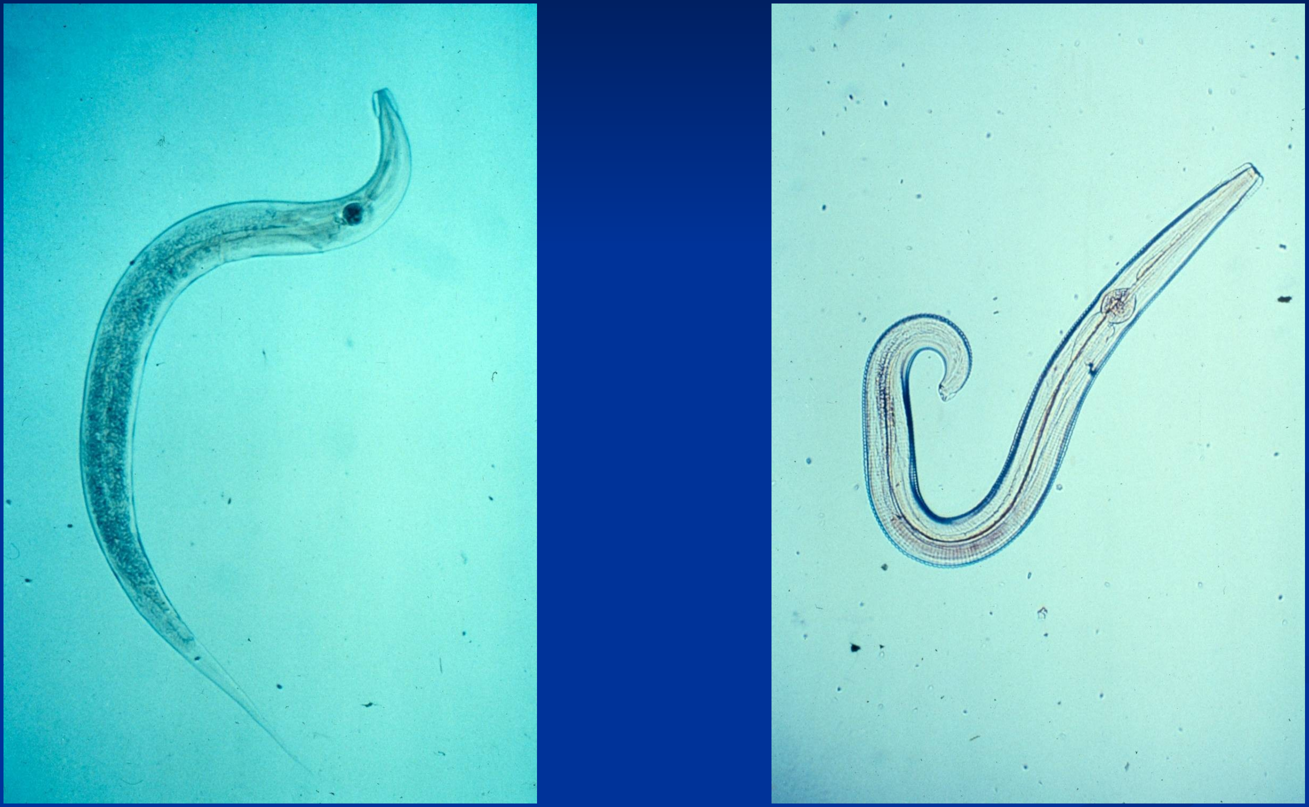
Pinworm Morphology

- Adults
  - Females 8-13 mm, long pointed tail
  - Males 0.3-0.5 mm, curved tail
  - Cephalic inflations, lateral alae, oxyurid esophagus (corpus, isthmus, bulb)
- Eggs
  - 50-60 x 20-30 µm
  - Flattened on one side
  - Infective in 4-6 hrs, viable for weeks
  - Resistant to desiccation

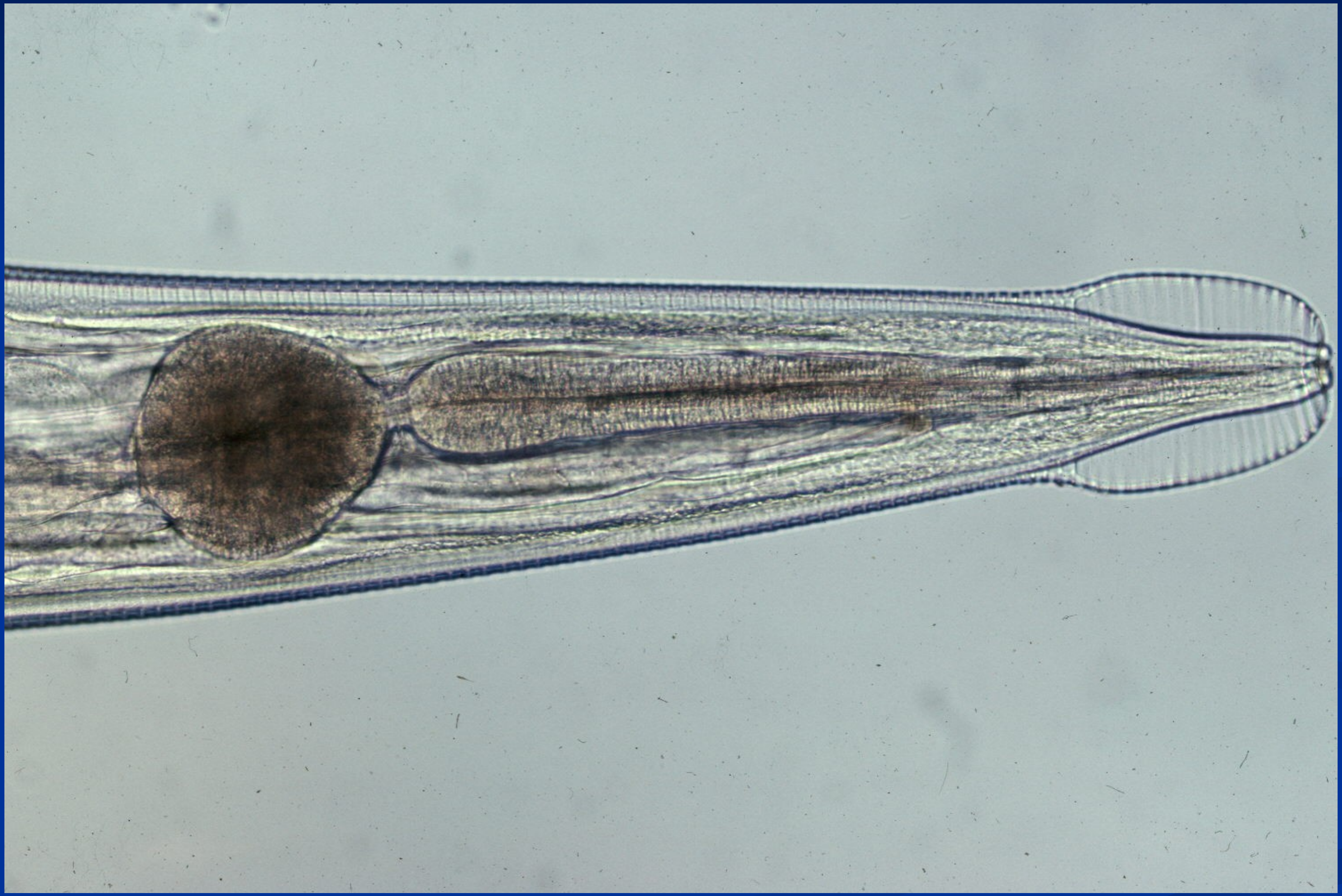
Pinworm adult females



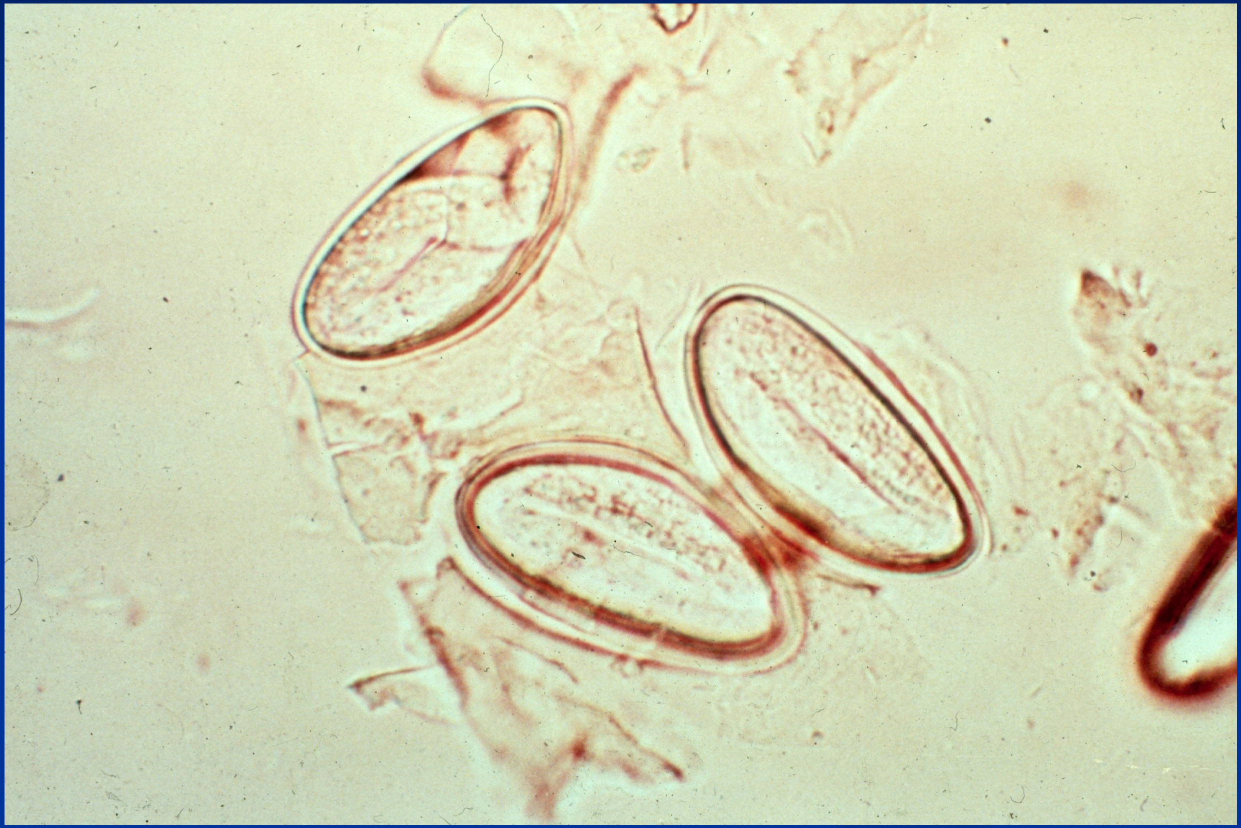
Pinworm female & male adults



Oxyurid esophagus & cephalic inflations



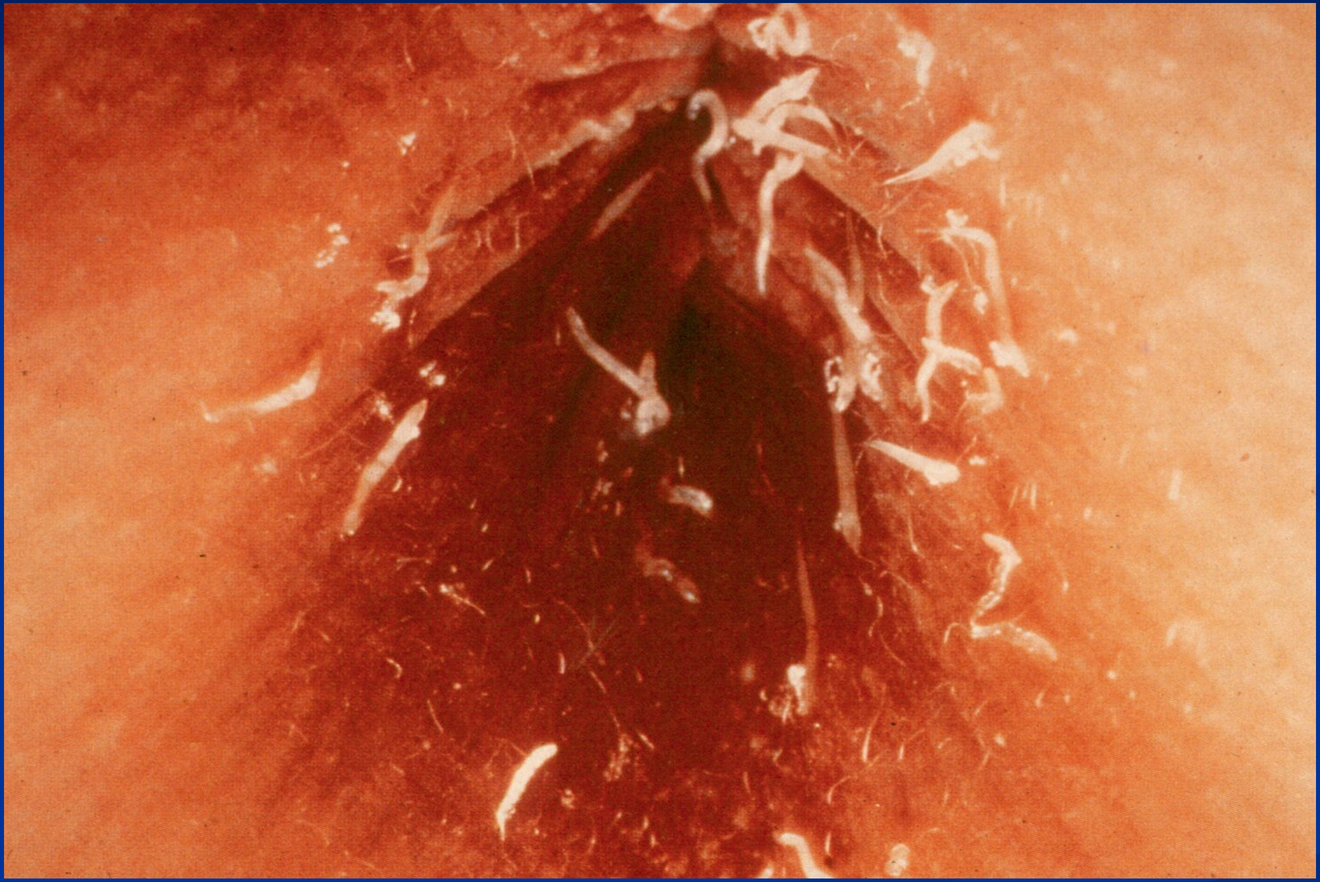
Pinworm eggs



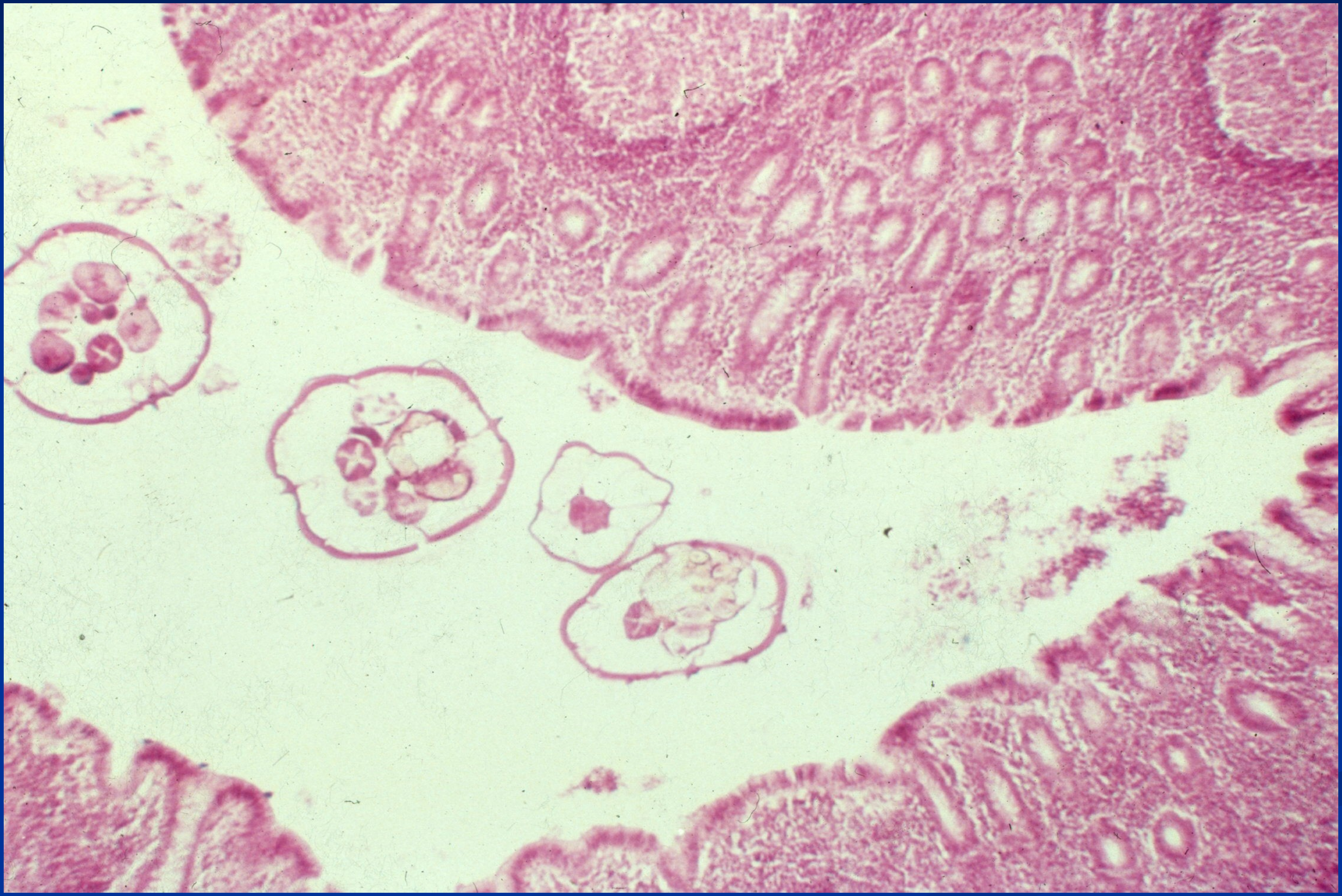
Pinworm Disease

- Perianal pruritis, insomnia, irritability, perianal lesions from scratching
- Tissue invasion rare
- Migration to ectopic sites can occur
  - Gravid females may migrate into vagina, uterus, fallopian tubes & peritoneal cavity
  - Appendix not uncommon site
- If ectopic, vaginitis, endometritis, salpingitis
- Significant eosinophilia with ectopic invasion

Pinworm females around anus



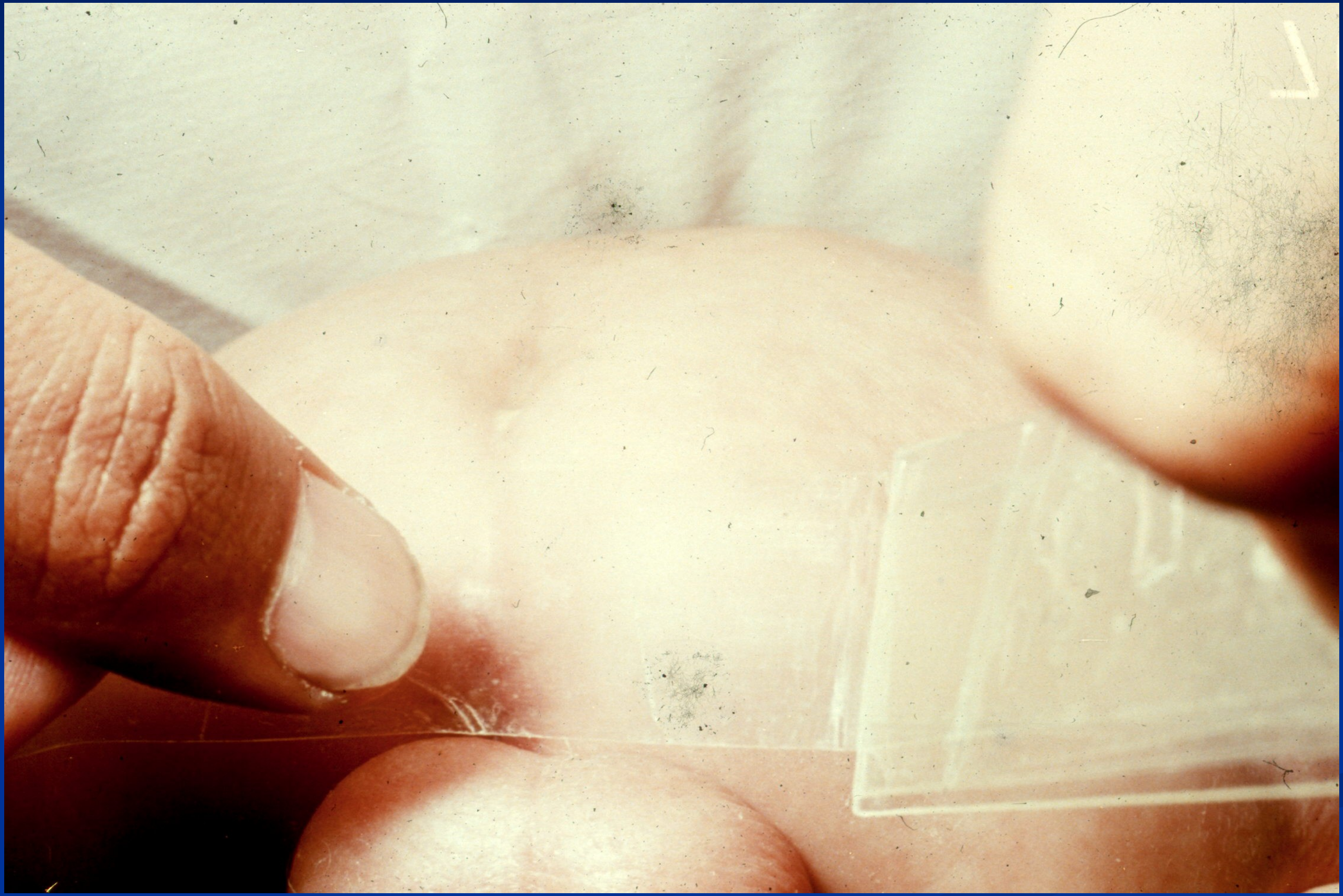
Pinworm in appendix



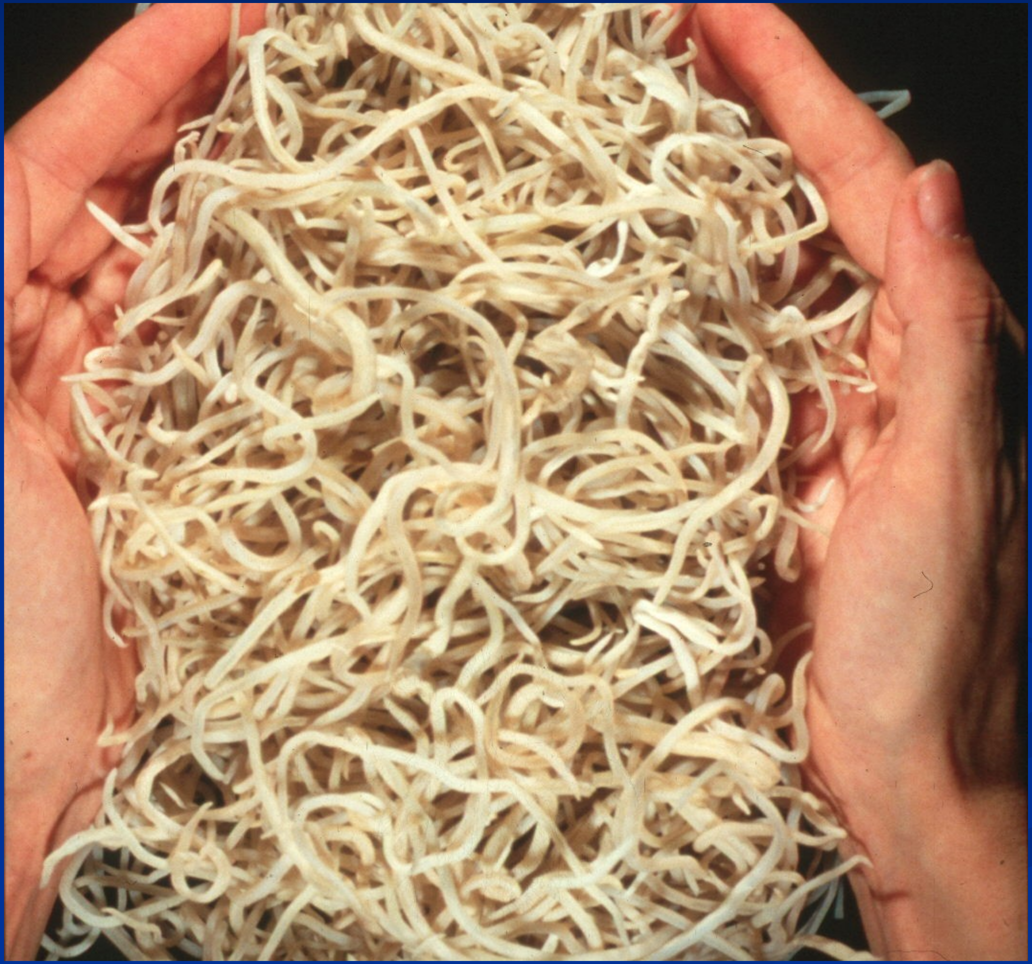
Pinworm Disease Dx & Tx

- Scotch tape technique method of choice
    - Picks up eggs in perianal area
    - Best in a.m. before toilet use or bathing
- Females seen around perianal area
- Eggs ***rarely*** found in stool, so O&P rarely useful
- Albendazole 400 mg po x 1x, repeat in 2 wks
- Mebendazole 100 mg po bid x 1x, repeat in 2 wks
- Pyrantel pamoate 11 mg/kg po daily x 1x, repeat q 2wks x 2

Scotch tape technique



Ascariasis
The Human Roundworm

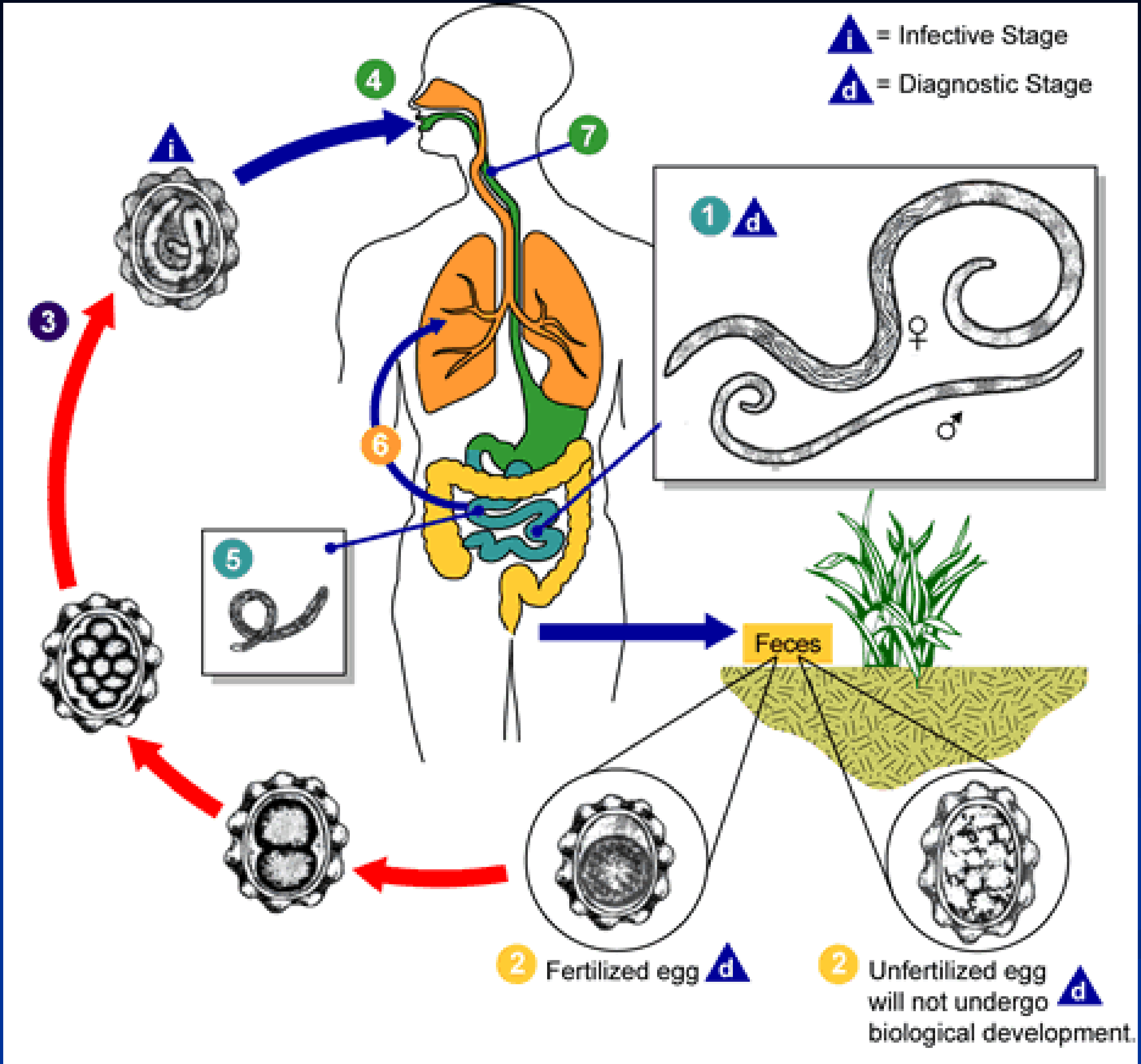


Ascariasis Epidemiology

- *Ascaris lumbricoides*
- Most prevalent helminthic infection
- 1.3 billion people infected
- Tropical and subtropical areas w/ moist soil or nightsoil use
- Prevalence
  - 71% in Asia
  - 13% in Latin America & Caribbean
  - 8% in Sub-Saharan Africa
- Heaviest worm burdens usually in children

Ascaris Life Cycle

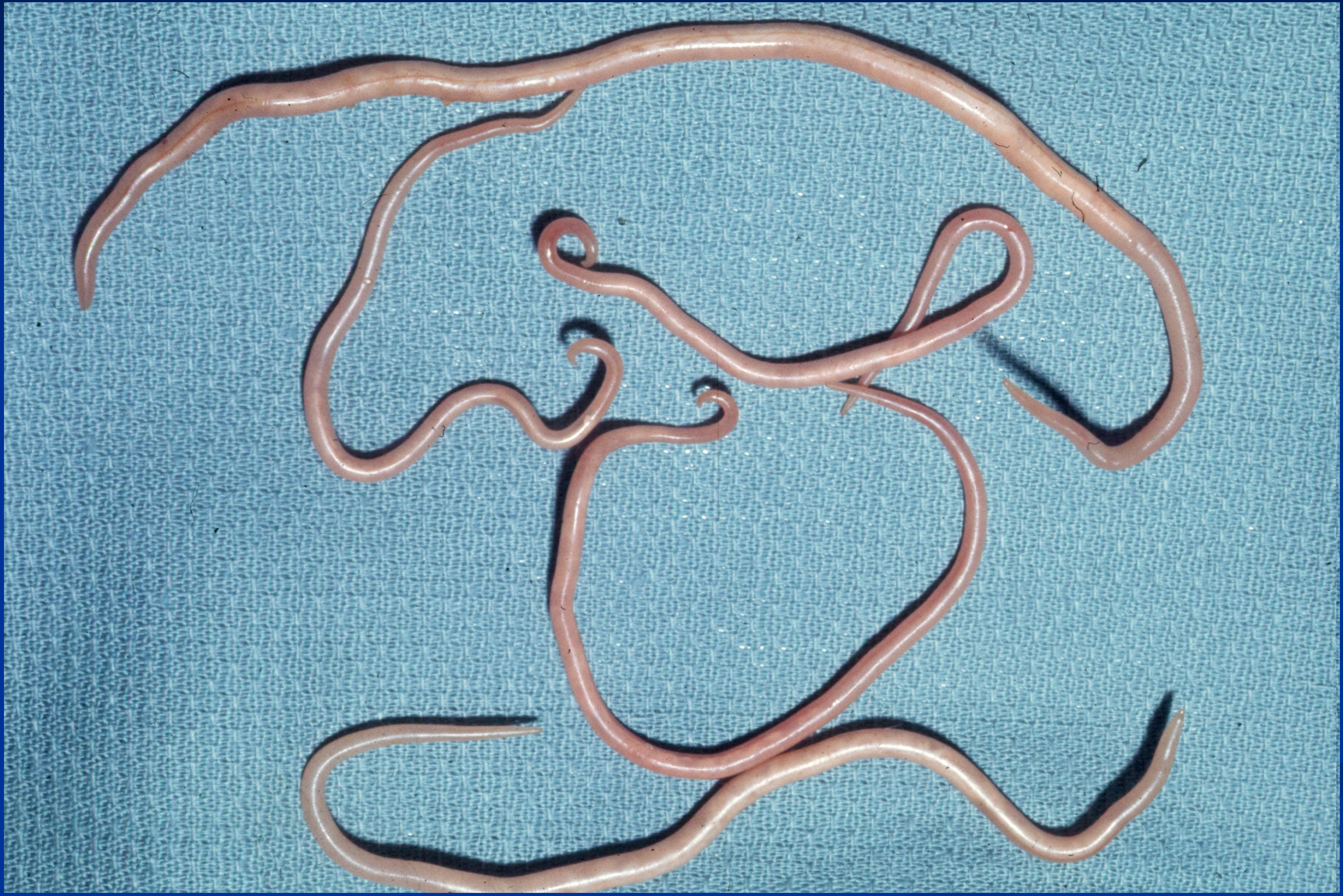
- Eggs deposited in soil & develop to infective 2nd stage larvae in 3 weeks
- Ingested by humans, larvae released in duodenum, penetrate mucosa & reach lungs via veins or lymph vessels, penetrate alveolar wall, molt 1-2x, migrate up trachea & down esophagus, mature & mate in ileum
- PP 2 months


i = Infective Stage
d = Diagnostic Stage
1 d
♀
♂
2 Fertilized egg d
2 Unfertilized egg will not undergo biological development. d
Feces
3
i
4
5
6
7

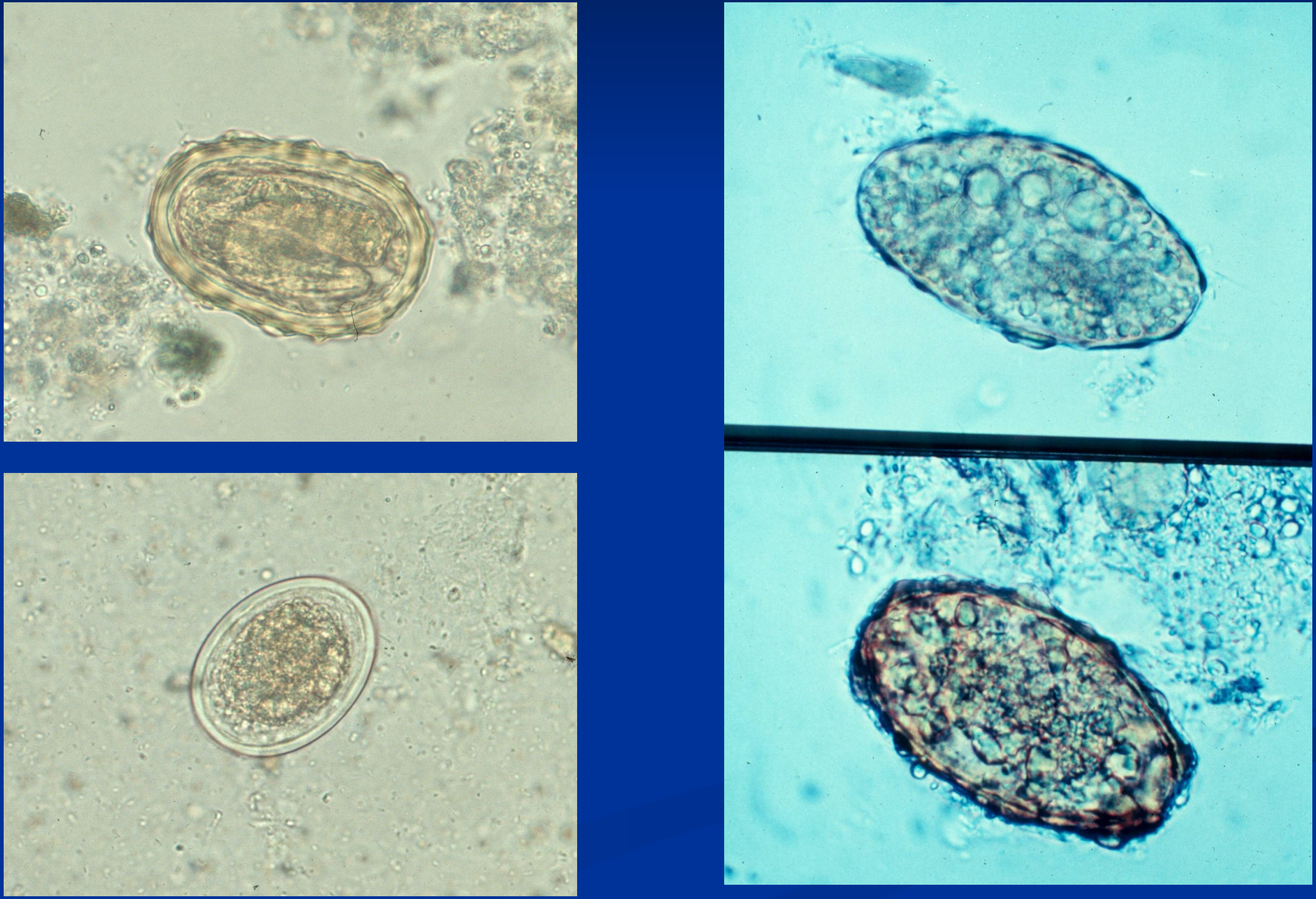
Ascaris Morphology

- Adults
  - Females 20-50 cm, have a girdle if unfertilized
  - Males 15-30 cm, curved tail
- Eggs
  - 45-70 x 35-50 µm
  - Mammillated outer layer
  - Decorticate eggs w/o mammillated layer still fertile
  - Infertile eggs 90 x 40 µm, sloppy mammillations

Ascaris adults

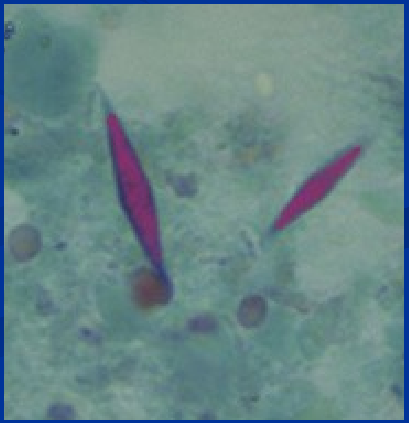


Ascaris eggs

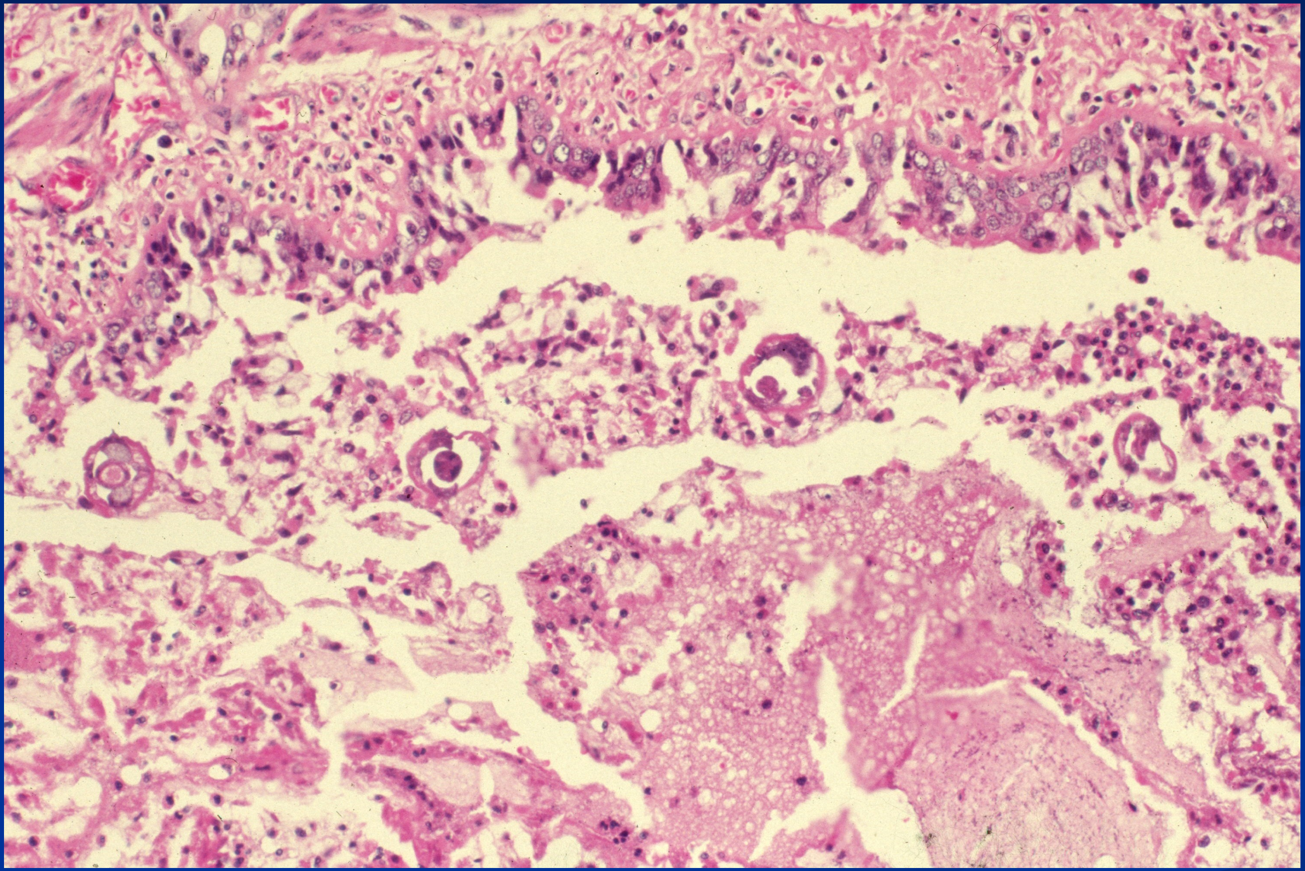


Ascariasis Pulmonary Phase

- 4-6 days post-infection, lasts 6-10 days
- Inflammatory response from migrating larvae
- Allergic rxns, bronchial spasms, granuloma formation
- Loeffler’s syndrome
  - Ascaris pneumonitis
  - Dyspnea, cough, wheezing, moderate fever, transient eosinophilia
  - Scattered shifting lung infiltrates on x-ray
  - Sputum contains eosinophils & Charcot-Leyden crystals



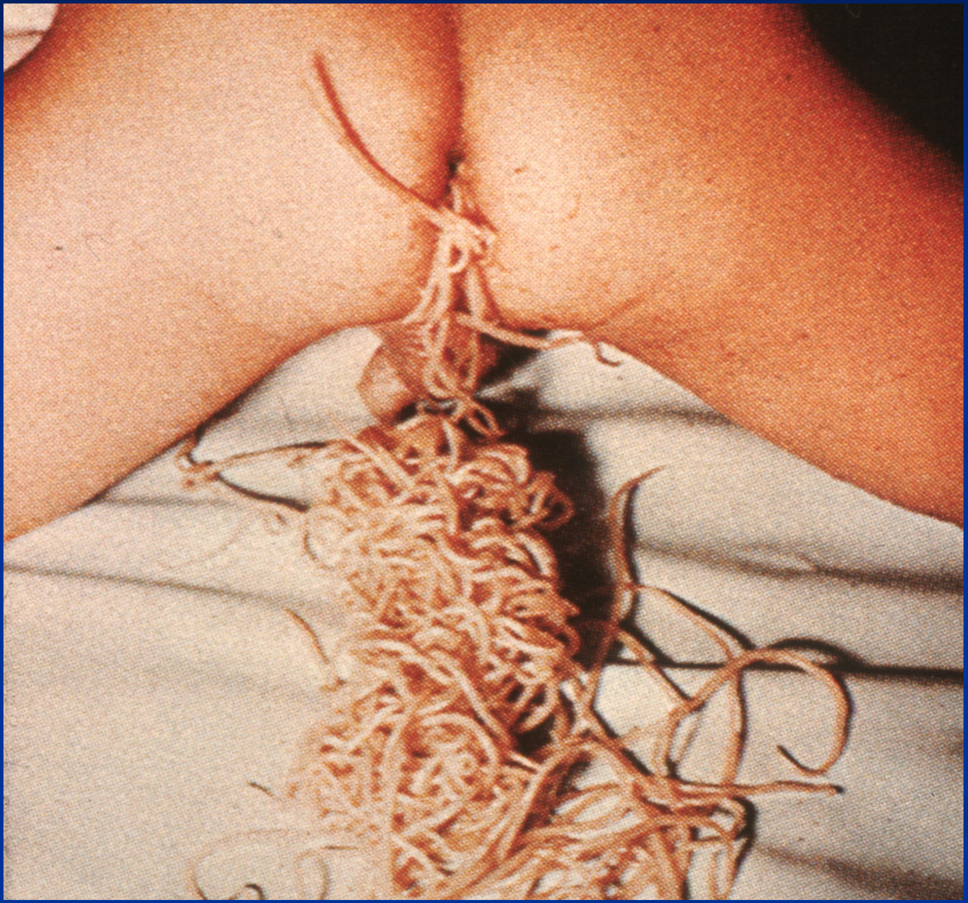
Ascaris larvae in lung



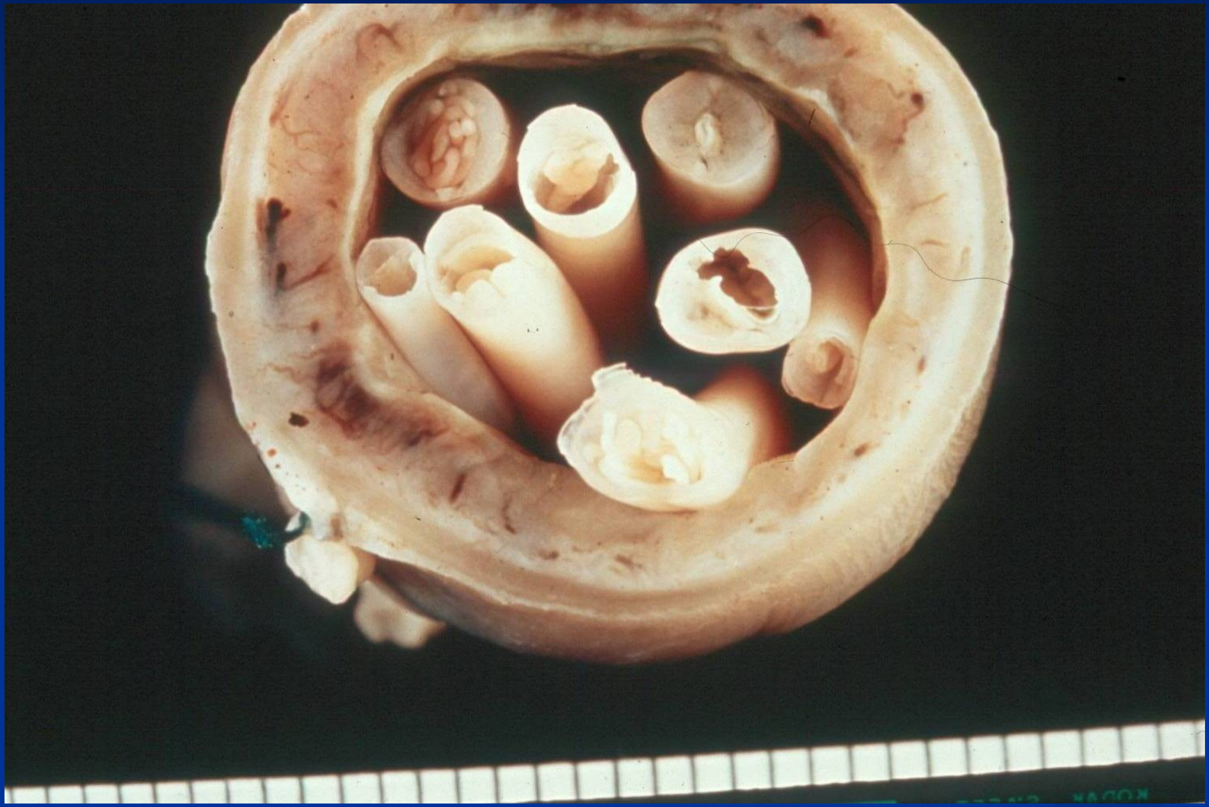
Ascariasis Intestinal Phase

- Adults in jejunum & ileum
- Large size + heavy worm burdens cause mechanical obstruction
- Nutritional impairment associated w/ burden
- Unmated females, fever, mebendazole tx, & anesthetics cause migration to ectopic sites
- Migrate to biliary & pancreatic ducts, liver, appendix
- Eggs can embolize to brain, heart, lungs
- Adults may spontaneously exit via mouth, nose, anus

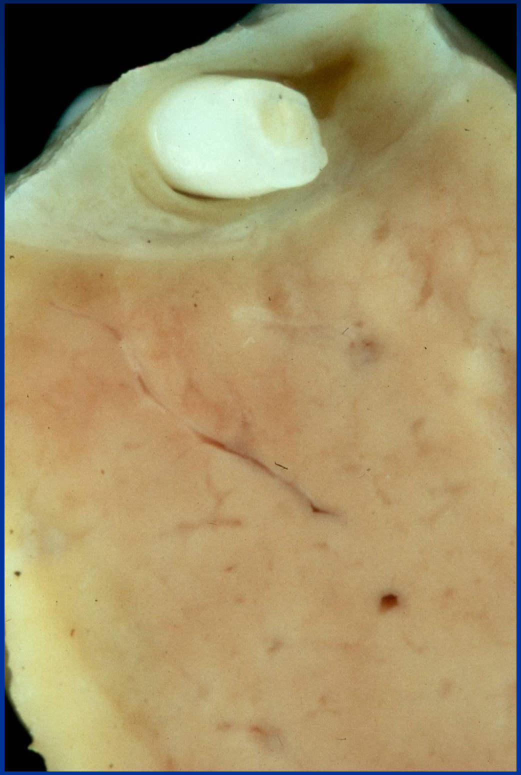
Bolus of ascarids



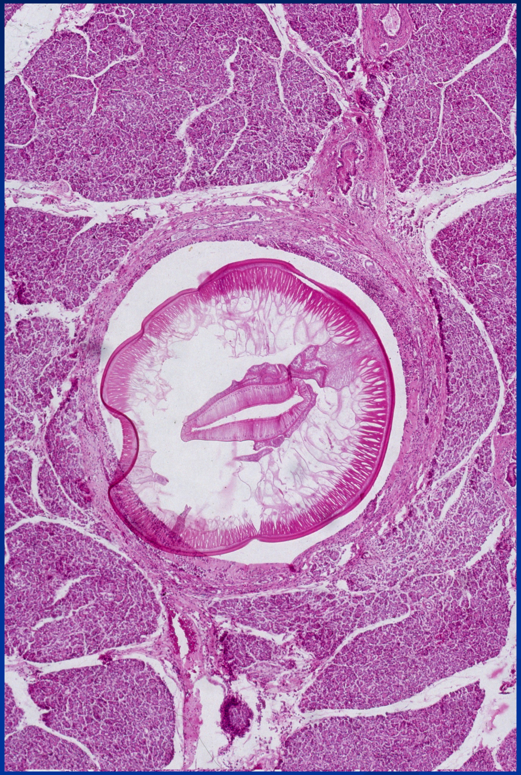
Intestinal obstruction



Ectopic locations

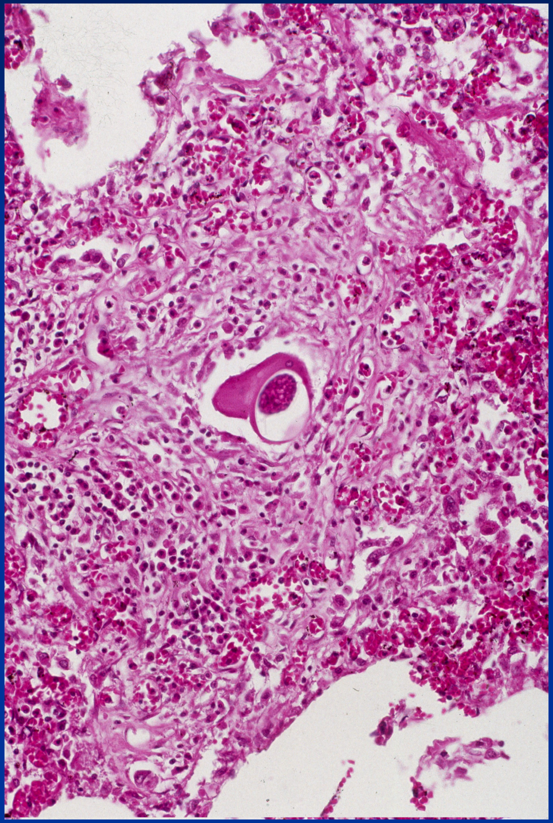


Adult in bile duct

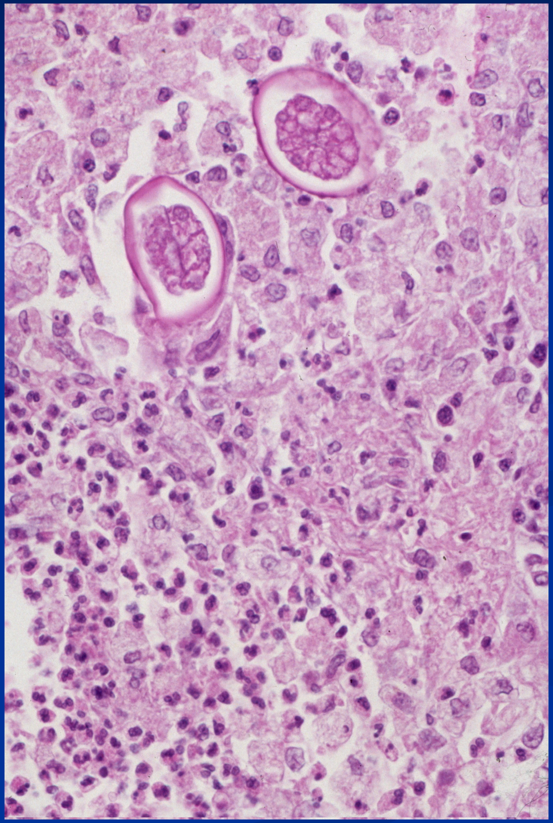


Adult in pancreatic duct

Ectopic locations



Egg in giant cell granuloma in lung



Eggs in liver

Ascariasis Diagnosis

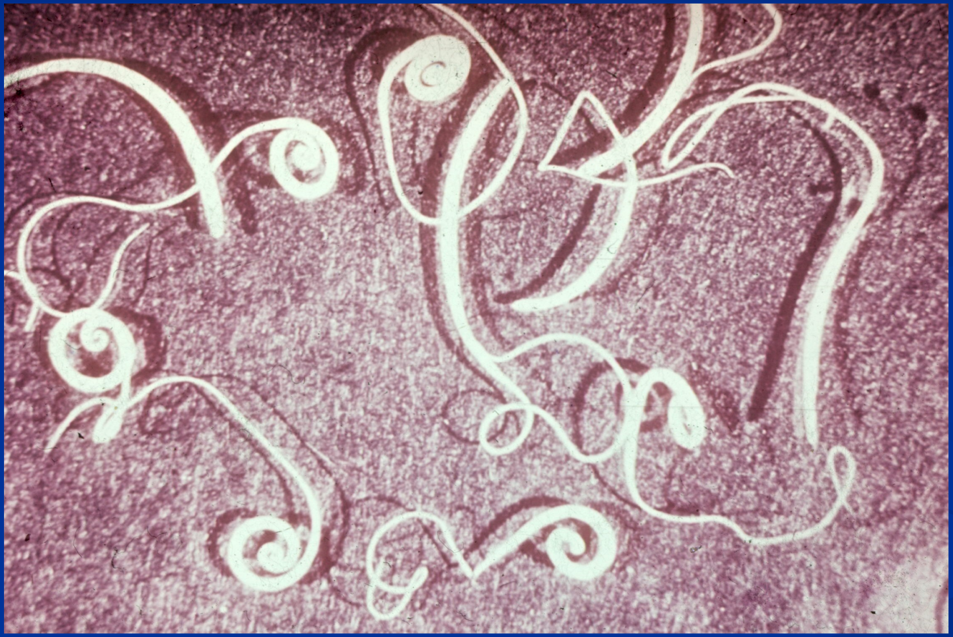
- Eggs in stool
  - Females produce approx. 200,000 eggs/day
- Larvae in sputum or gastric washings
- Adult emerging from orifice

Ascariasis Treatment

- Albendazole 400 mg po for 1 dose
- Mebendazole 100 mg po bid x 3d
- Pyrantel pamoate 11 mg/kg po for 1 dose
- For obstruction, piperazine citrate 75 mg/kg daily x 2d

Trichuriasis

Whipworm

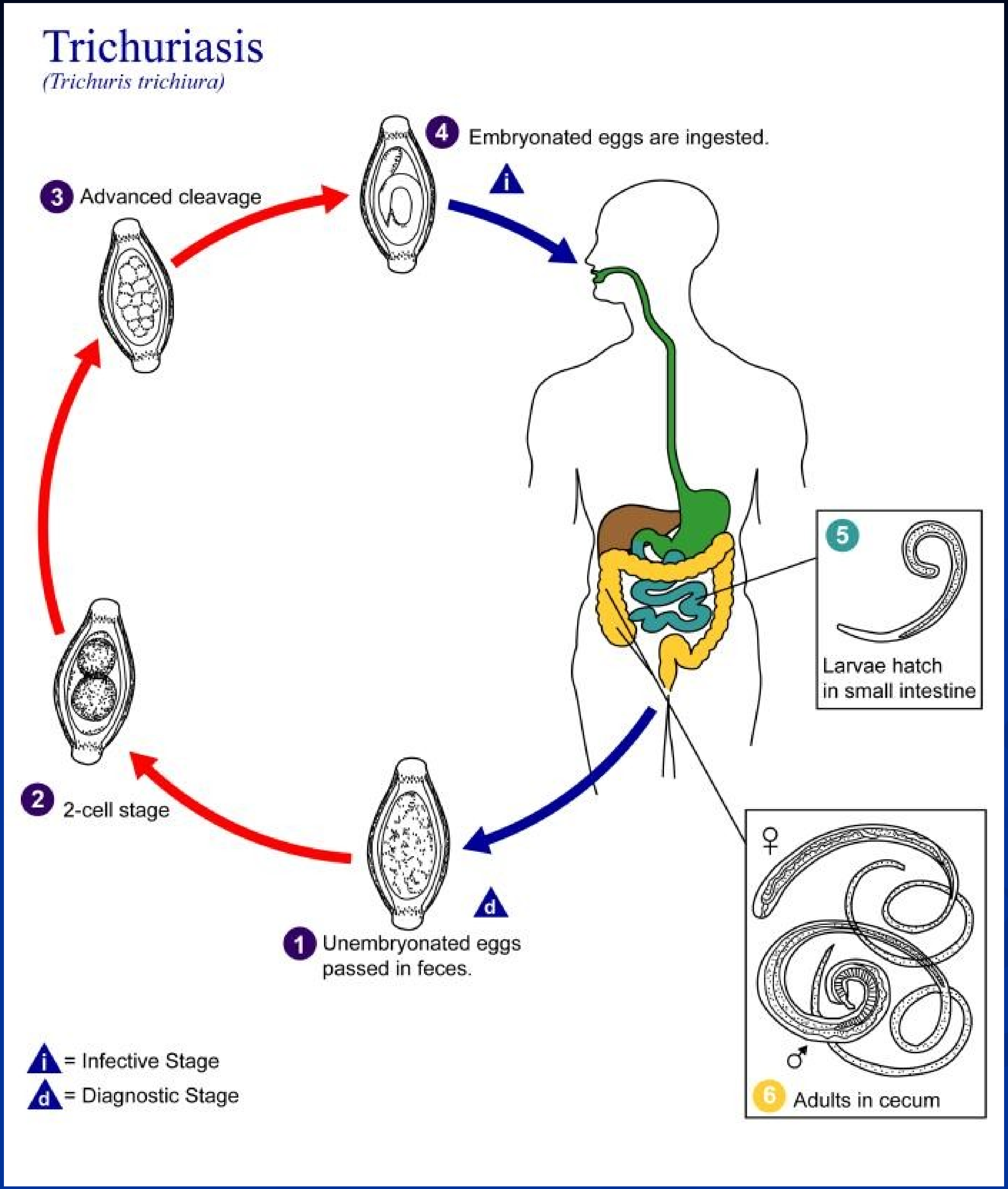


Trichuriasis Epidemiology

- *Trichuris trichiura*
- 900 million people infected
- Global distribution
- Less resistant to drying than *Ascaris*
- Heaviest burdens in children
- Humans primary host, however it’s also been reported from pigs

Trichuriasis Life Cycle

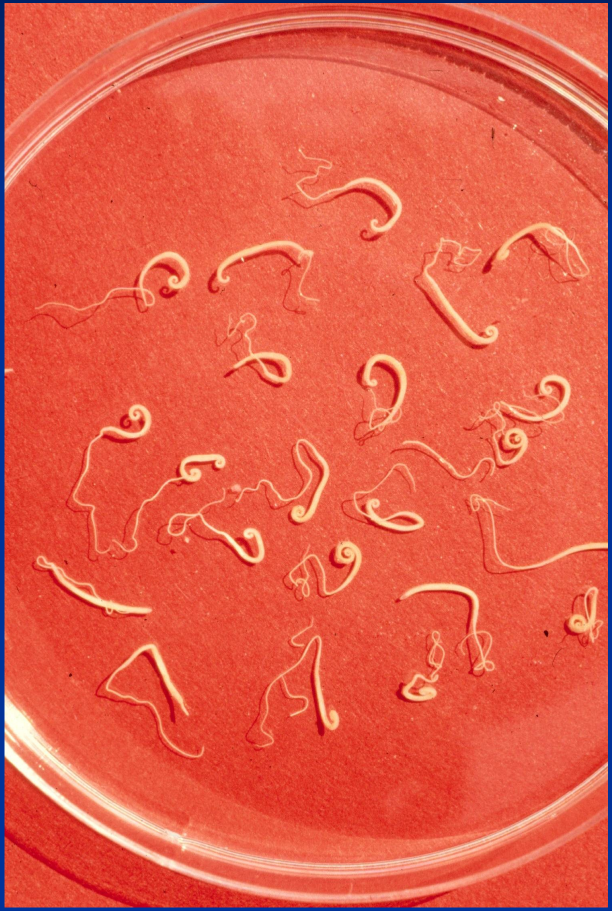
- Unembryonated eggs passed in stool
- Egg contains infective 1st stage larvae in 3 weeks which is swallowed by human
- Hatches, matures, threads anterior portion of body through colon epithelium
- Eggs laid and passed out in feces
- PP 1-3 months

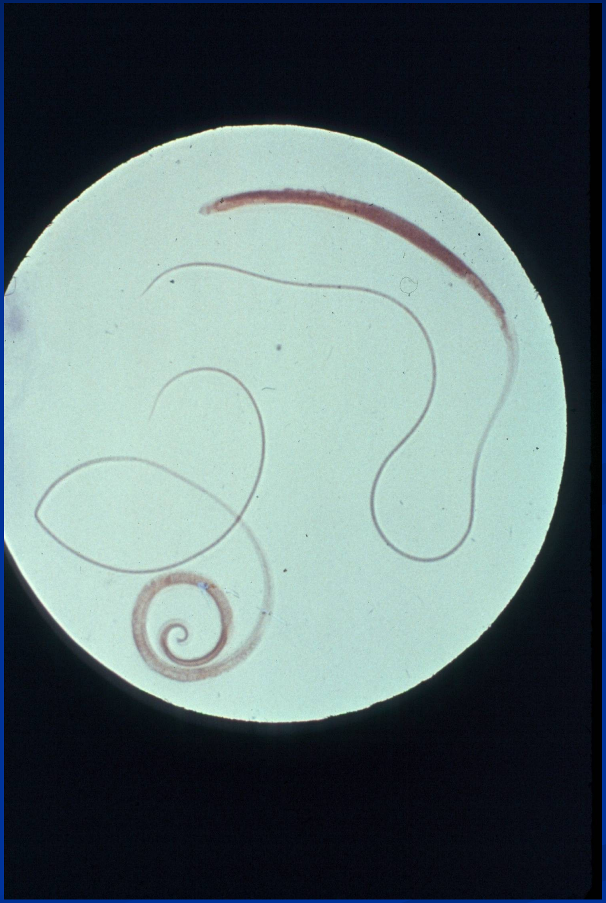

Trichuriasis
(Trichuris trichiura)
4 Embryonated eggs are ingested.
i
3 Advanced cleavage
5
Larvae hatch in small intestine
2 2-cell stage
♀
♂
6 Adults in cecum
d
1 Unembryonated eggs passed in feces.
i = Infective Stage
d = Diagnostic Stage

Trichuriasis Morphology

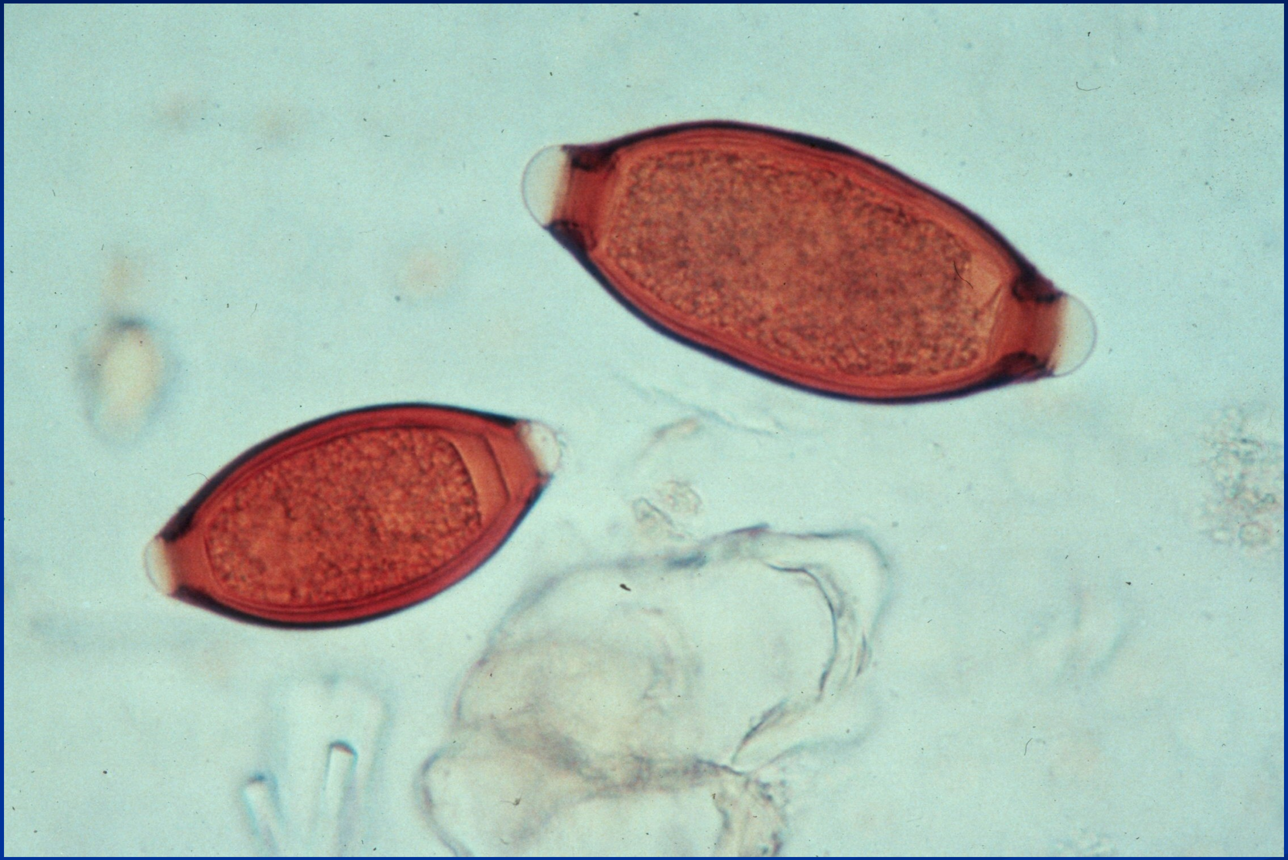
- Adults
  - Whip like anterior & thick posterior end
  - Females 30-50 mm
  - Males 30-45 mm, coiled tail
- Eggs
  - 52 x 22 µm
  - Dark thick shell
  - Bipolar mucoid plugs

Trichuris adults





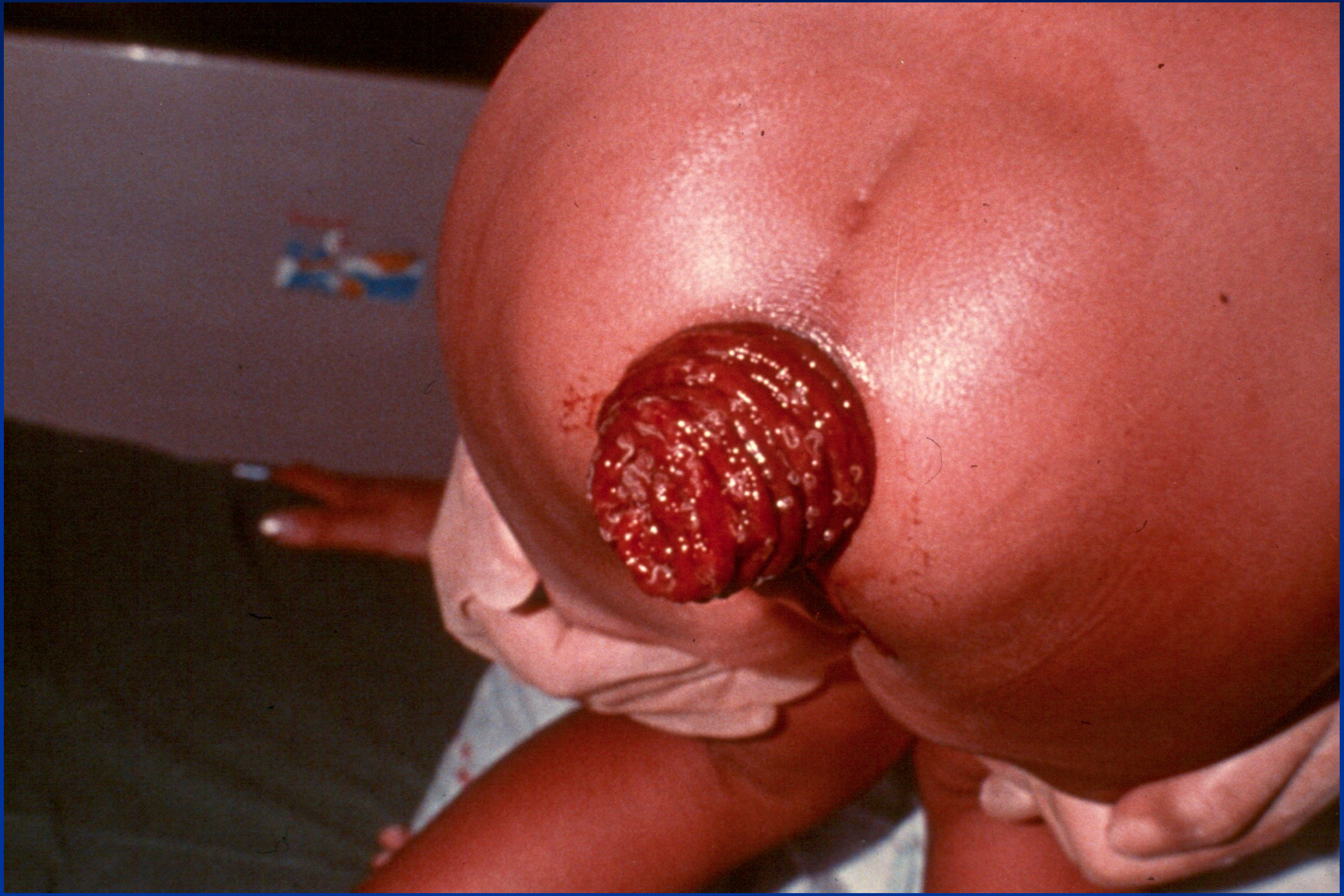
Trichuris eggs



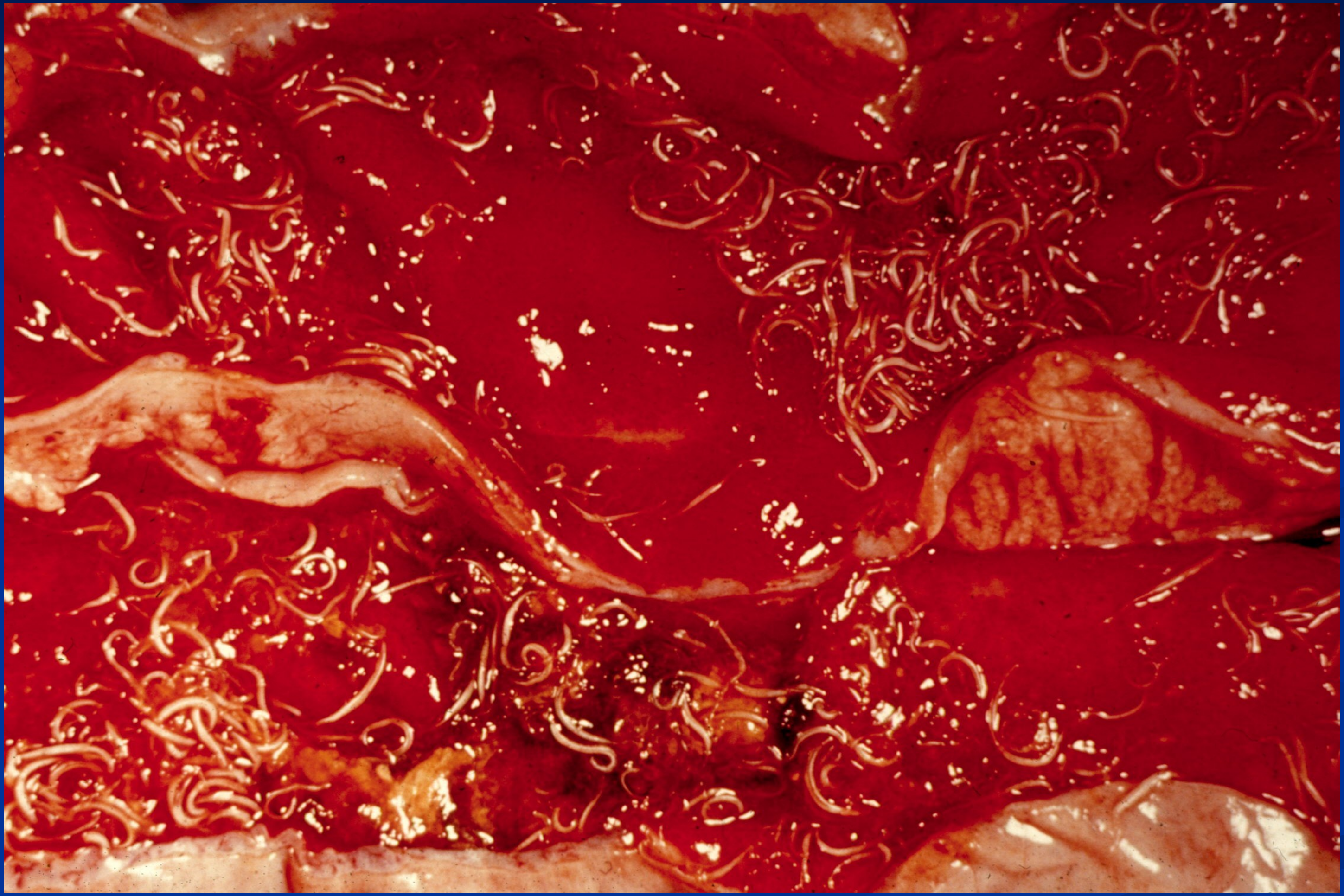
Trichuriasis Disease

- Pathology associated w/heavy infections
- Hemorrhage, mucosal cell destruction, inflammatory infiltrates
- Abdominal pain, diarrhea, distention, flatulence, anorexia, weight loss
- In heavy infections, profuse bloody diarrhea, tenesmus, rectal prolapse
- May invade appendix

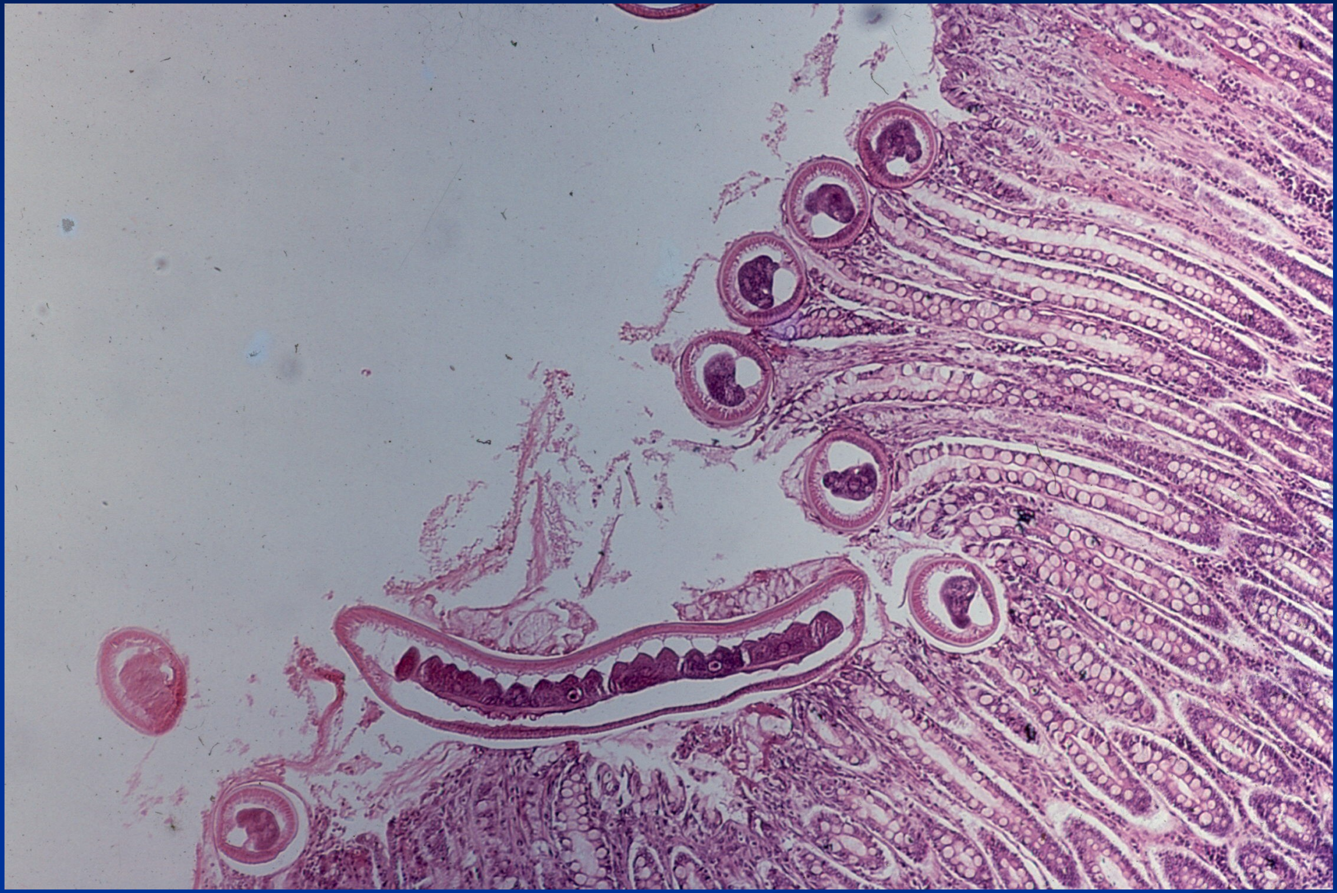
Rectal prolapse



Adults in situ



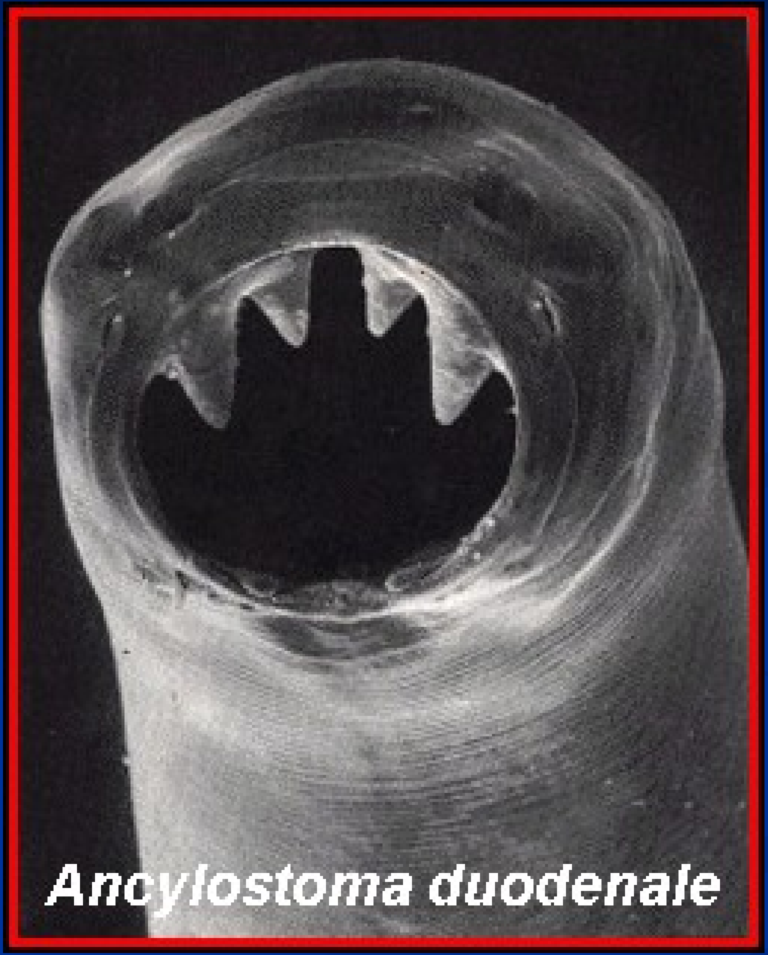
Adult threaded through mucosal epithelium



Trichuriasis Dx & Tx

- Eggs in stool
- Proctoscopy
  - Hyperemic & edematous mucosa
  - Adults worms visible
- Eosinophilia 5-15% in early stage
- Iron deficient anemia
- Albendazole 400 mg po for 1 dose or mebendazole 100 mg po bid x 3d

Hookworms



Ancylostoma duodenale

Hookworm Epidemiology

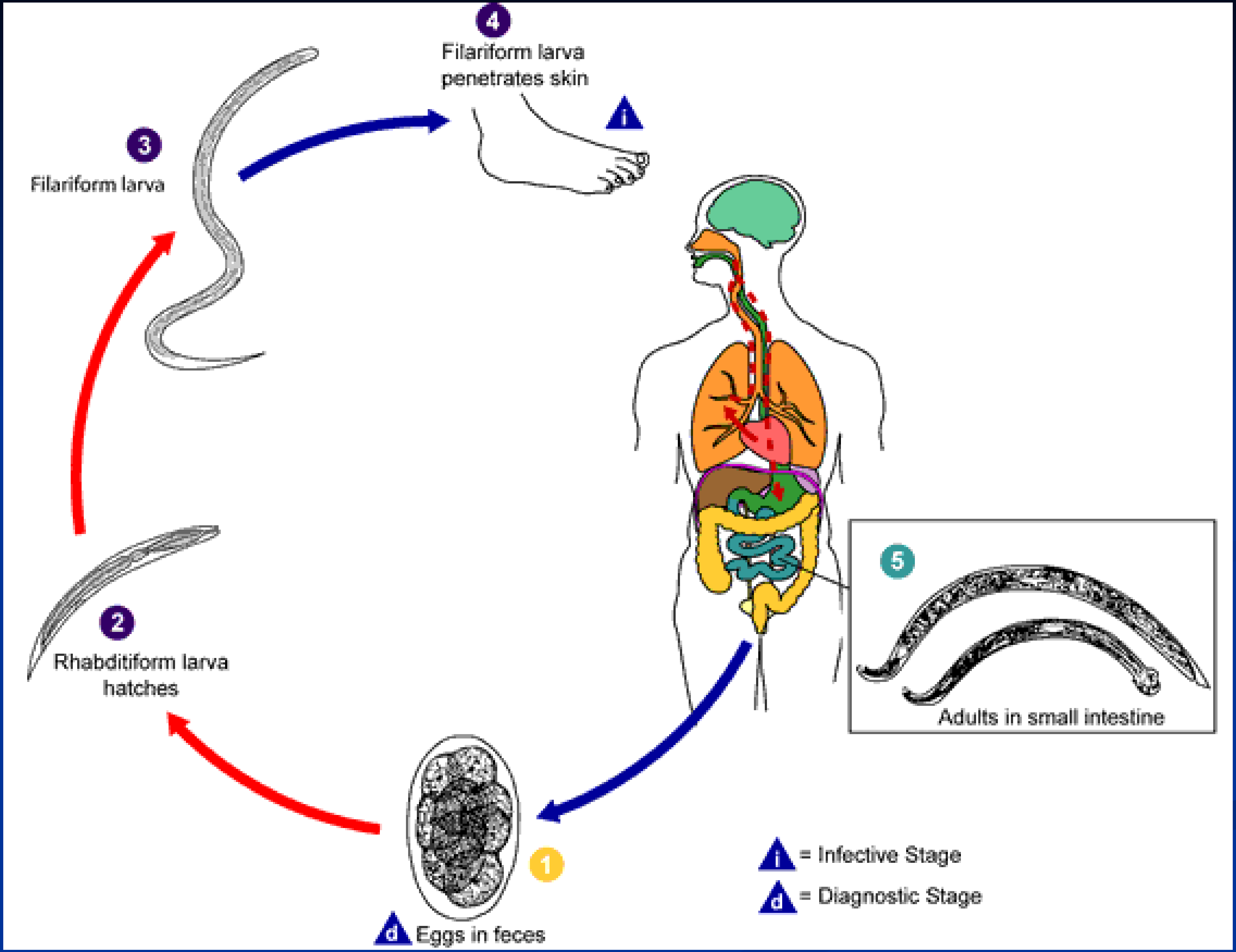
- 1 billion people infected
- Associated w/ areas of promiscuous defecation & nightsoil use
- Humans only reservoir
- Prevalence up to 90% in endemic areas
- Worldwide distribution

Human Hookworm Species

- *Necator americanus* - New World Hookworm
  - Cutting plates
  - Skin penetration only
  - Daily blood loss/worm 0.03-0.25 ml
  - S US, Caribbean, Latin America, C & S Africa, Asia, Pacific
- *Ancylostoma duodenale* - Old World Hookworm
  - Cutting teeth
  - Can infect orally as well as via skin penetration
  - Daily blood loss/worm 0.1-0.5 ml
  - S Europe, N coast of Africa, N China, Japan, India, SE Asia, Pacific

Hookworm Life Cycle

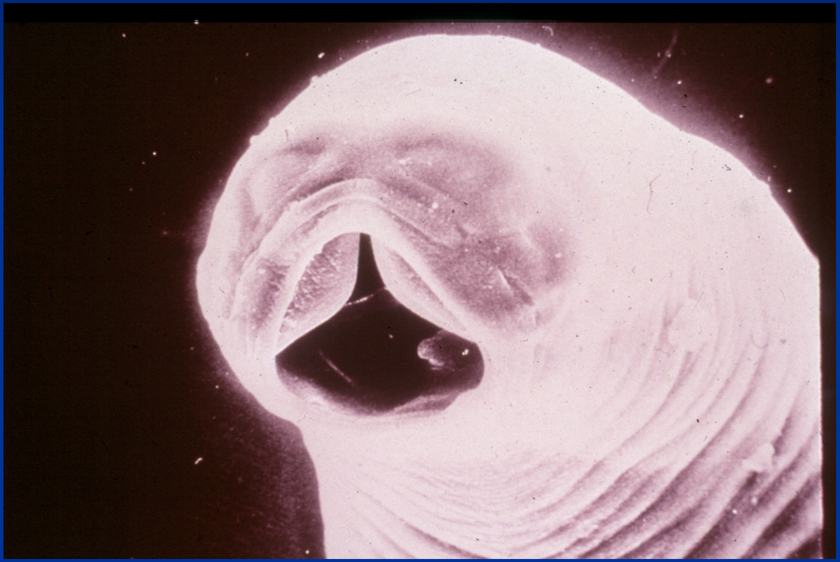
- Eggs passed in stool, 1st stage larvae hatch, molt to infective 3rd stage in 7-9 days
- Penetrate skin (also infect orally w/ *Ancylostoma*), enter venules, pass through heart to lungs, break out of capillaries, migrate up & are swallowed
- Oral *Ancylostoma* bypasses lung migration
- Attach to intestinal mucosa, mature, mate, produce eggs
- PP 5 wks


4
Filariform larva penetrates skin
i
3
Filariform larva
2
Rhabditiform larva hatches
5
Adults in small intestine
1
d Eggs in feces
i = Infective Stage
d = Diagnostic Stage

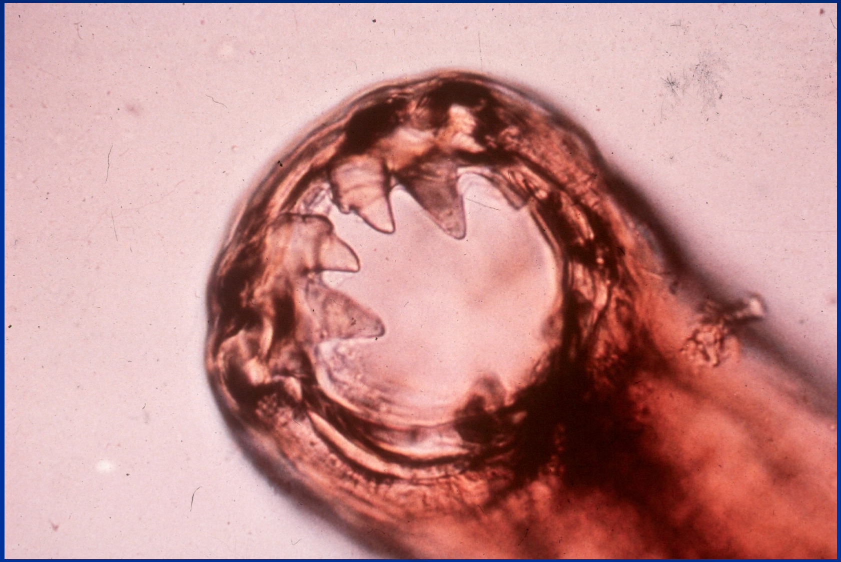
Hookworm Morphology

- Eggs
  - Thin colorless shell
  - Embryo in early cleavage
  - 60-75 x 35-40 µm
- 1st stage larva - rhabditoid
  - 250-300 µm
  - Long buccal capsule
  - Small genital primordium
- 3rd stage infective larva - filariform
  - 500-600 µm
  - 1:3 esophagus to intestine ratio
  - Pointed tail

Hookworm adults

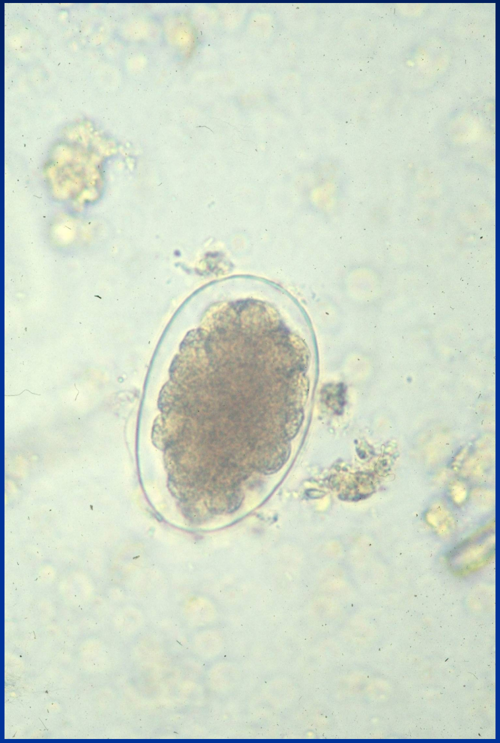


Necator americanus

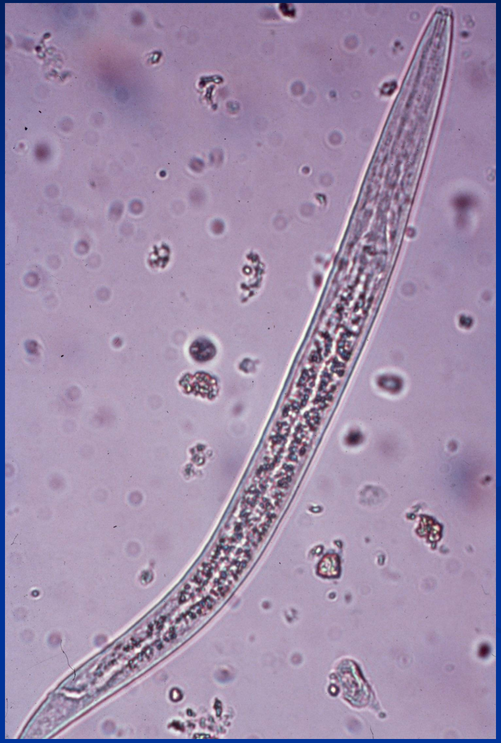


Ancylostoma duodenale

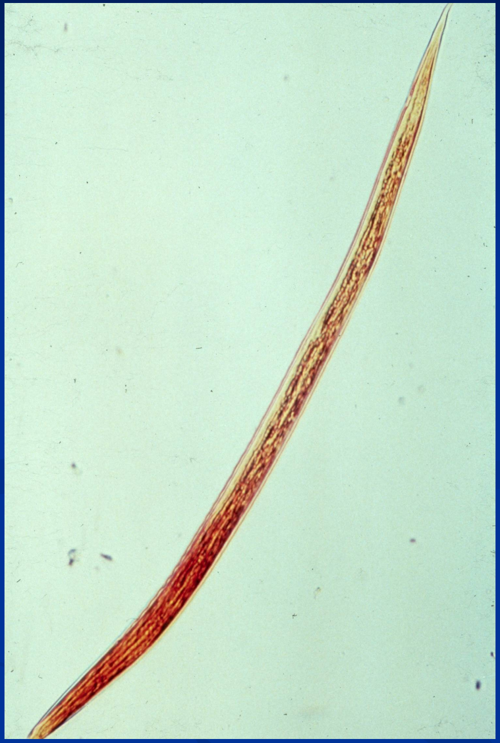
Hookworm egg & larvae



Egg



Rhabditoid larva

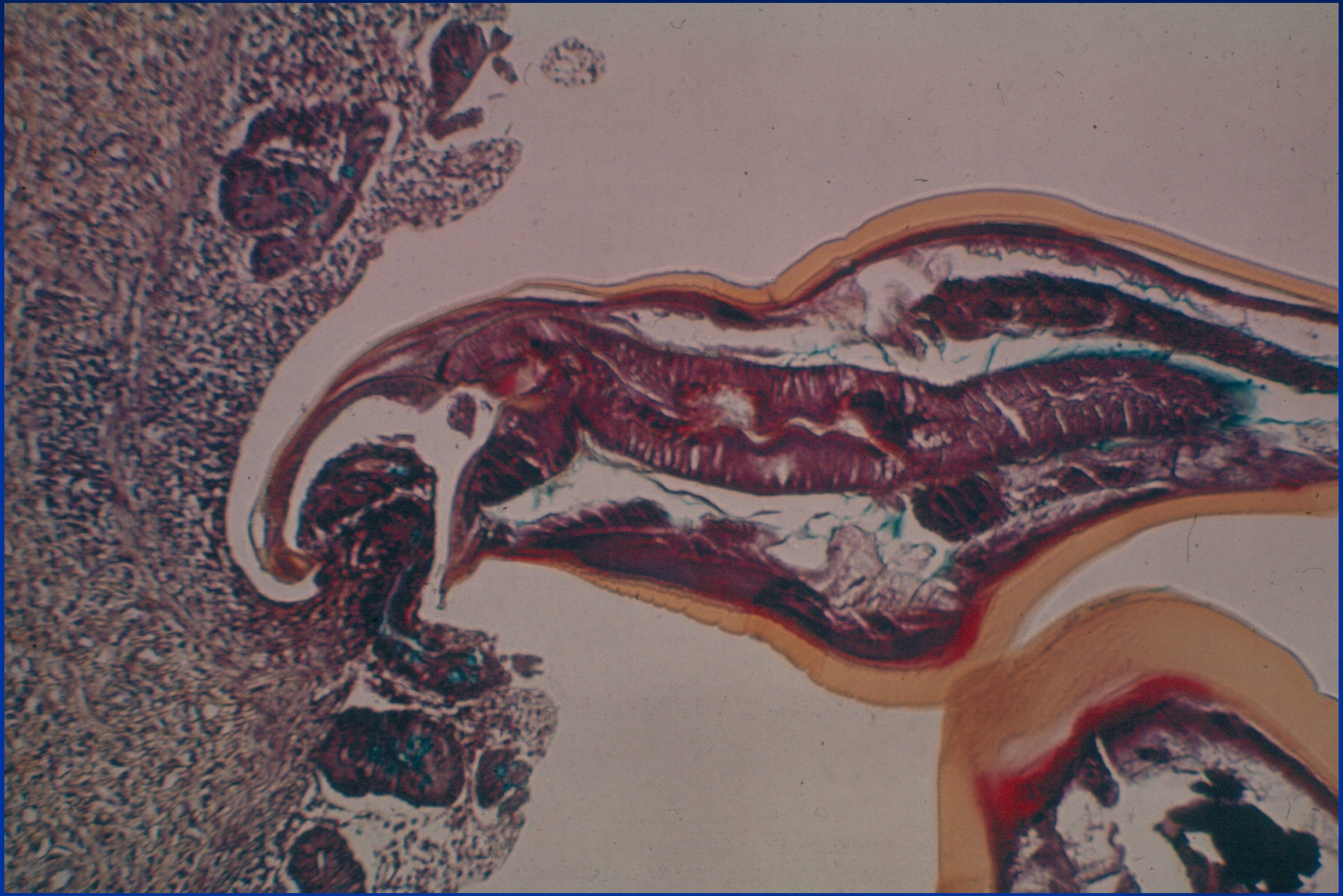


Filariform larva

Hookworm Disease

- Cutaneous phase
  - Erythema, pruritis, papulovesicular eruption
- Pulmonary phase
  - Usually asymptomatic
  - Dry cough, wheezing, fever, blood in sputum
  - Eosinophilia 30-60%
- Intestinal phase
  - Iron deficiency anemia
  - Diarrhea, pica, anorexia, dyspnea, growth retardation

Hookworm feeding (movat 25x)



Hookworm Disease Dx & Tx

- Eggs in stool
- Concentration methods
- Charcoal stool culture
- Anemia, occult blood, & eosinophilia
- Albendazole 400 mg po for 1 dose
- Mebendazole 100 mg po bid x 3d
- Pyrantel pamoate 11 mg/kg po daily x 3d

Strongyloidiasis

Strongyloidiasis Epidemiology

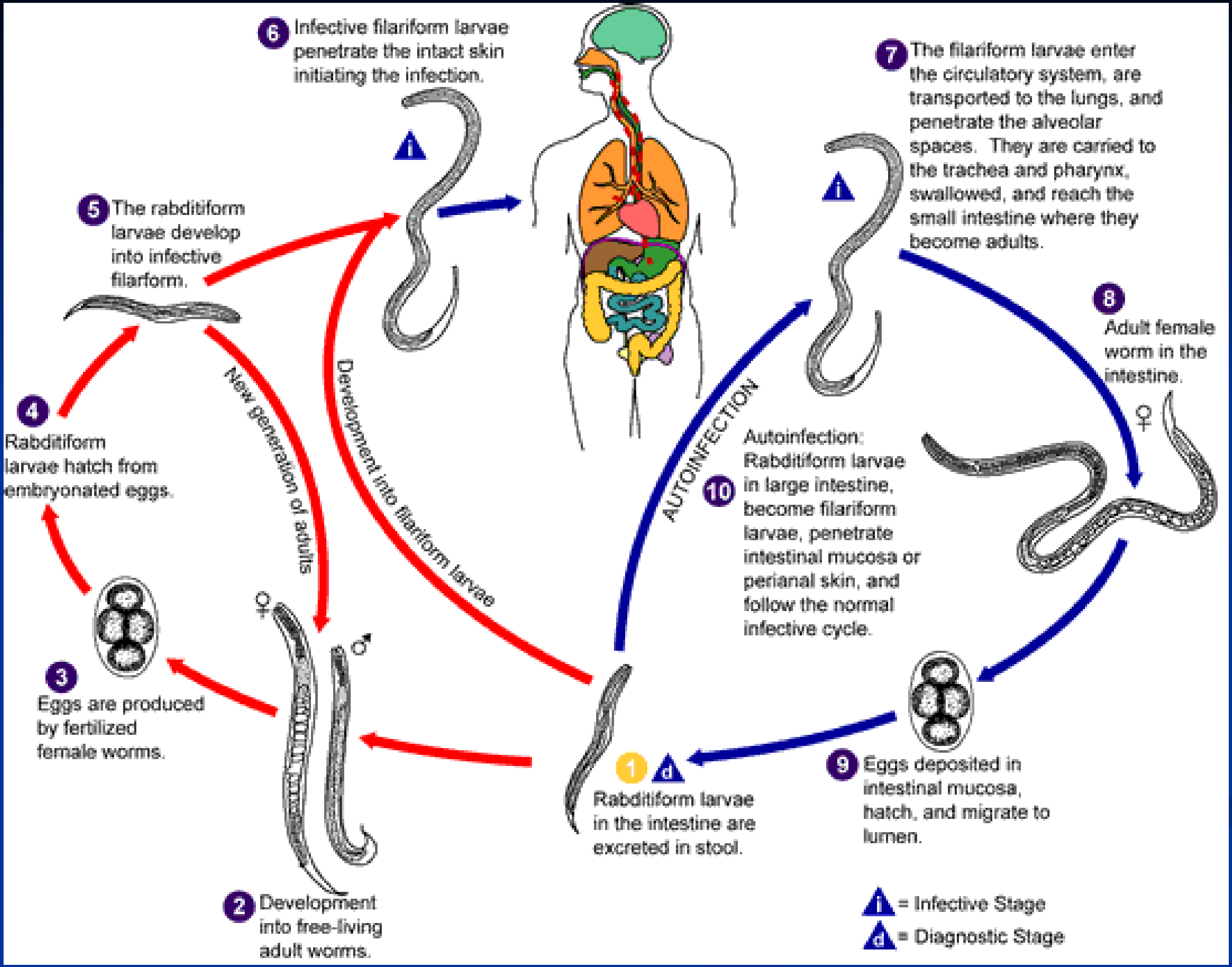
- *Strongyloides stercoralis* usual culprit
  - *S. fuelleborni* in African primates & humans, similar species in humans in PNG
- Worldwide in tropics & subtropics, up to 30% infection rate in some endemic areas, approx. 50-100 million infected
- Endemic in tropical Asia, Africa, Latin America, southern US, southern & eastern Europe
- Hyperinfection in humans via entire life cycle capable of occurring in human host, intermediate host not necessary

Life Cycle

- First stage rhabditoid larvae shed in feces
- Molt 4 times to adulthood, mate, produce eggs
- However, some female 1st stage larvae will molt to third stage filariform larvae & become infective to humans
- These penetrate skin, enter vessels, pass to lungs, break into alveoli, migrate to intestine, invade mucosal epithelium of duodenum & jejunum, mature & lay eggs; no male needed

Life Cycle (cont)

- Normally eggs hatch in gut, 1st stage larvae excreted in stool
- Autoinfection
  - 1st stage larvae molt to 3rd stage in transit through intestine, invade mucosa of ileum or colon, pass through lungs to intestine, mature as females to produce more larvae
  - Can lead to hyperinfection w/ enormous numbers of larvae, particularly if host is immunosuppressed



① d Rabditiform larvae in the intestine are excreted in stool.

② Development into free-living adult worms.

③ Eggs are produced by fertilized female worms.

④ Rabditiform larvae hatch from embryonated eggs.

⑤ The rabditiform larvae develop into infective filarform.

⑥ Infective filariform larvae penetrate the intact skin initiating the infection.

⑦ The filariform larvae enter the circulatory system, are transported to the lungs, and penetrate the alveolar spaces. They are carried to the trachea and pharynx, swallowed, and reach the small intestine where they become adults.

⑧ Adult female worm in the intestine.

⑨ Eggs deposited in intestinal mucosa, hatch, and migrate to lumen.

⑩ Autoinfection: Rabditiform larvae in large intestine, become filariform larvae, penetrate intestinal mucosa or perianal skin, and follow the normal infective cycle.

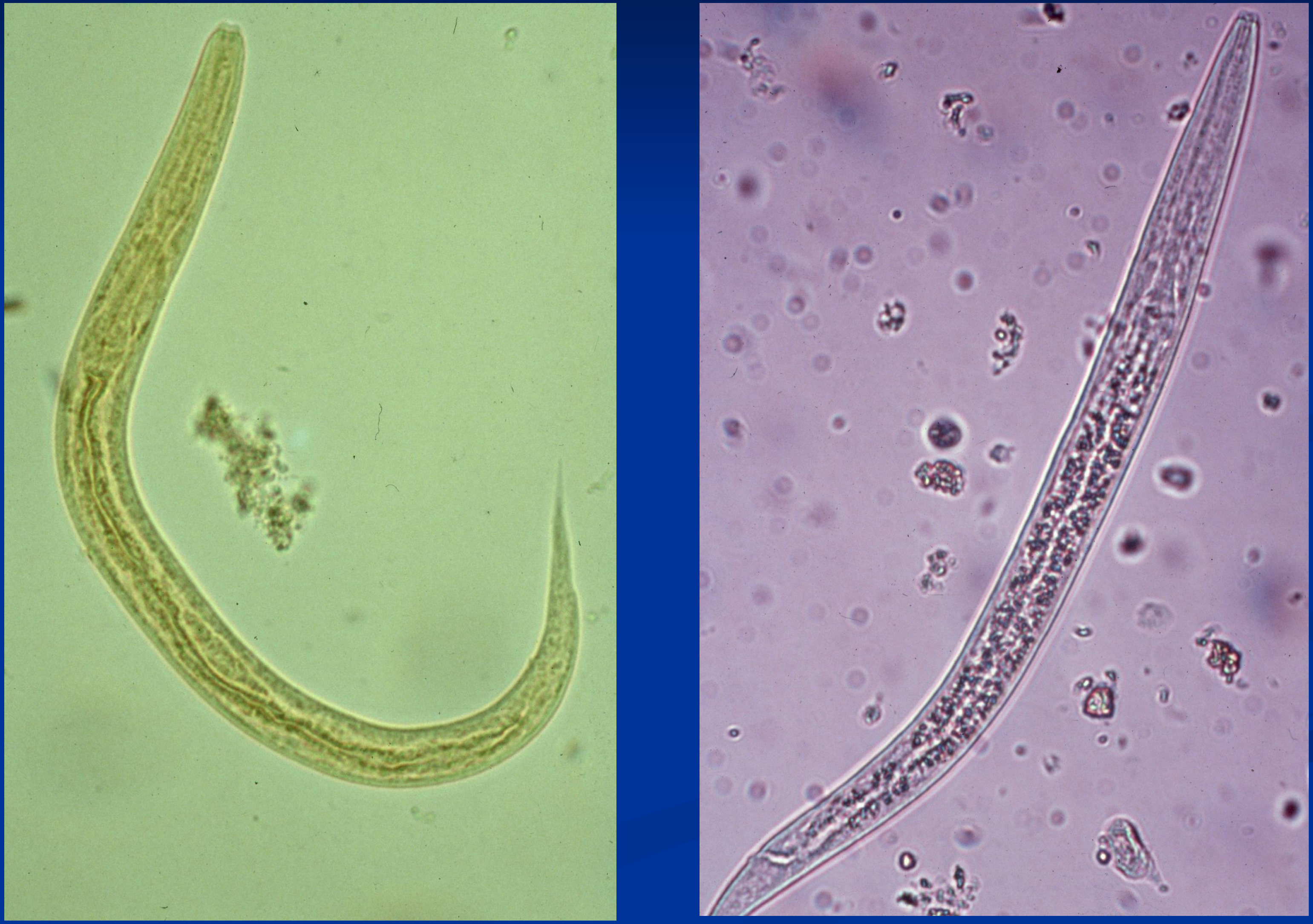
i = Infective Stage
d = Diagnostic Stage

Larval morphology

	Strongyloides		Hookworm	
	Rhabditoid (L1)	Filariform (L3)	Rhabditoid (L1)	Filariform (L3)
BC	Short		Long	
GP	Prominent		Indescript	
Esoph:Int ratio		1:1		1:3
Tail		Notched		Pointed

BC = buccal capsule GP = genital primordium

Strongyloides vs Hookworm L1

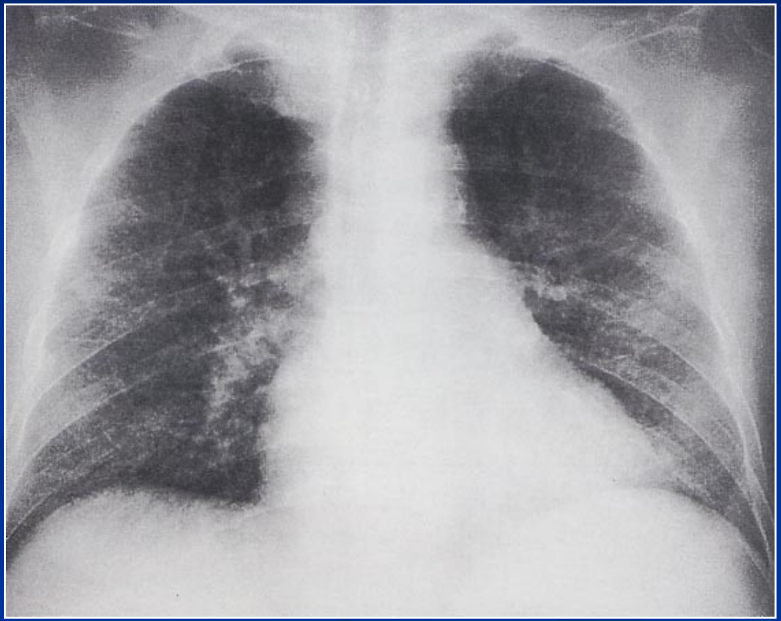


Cutaneous Phase

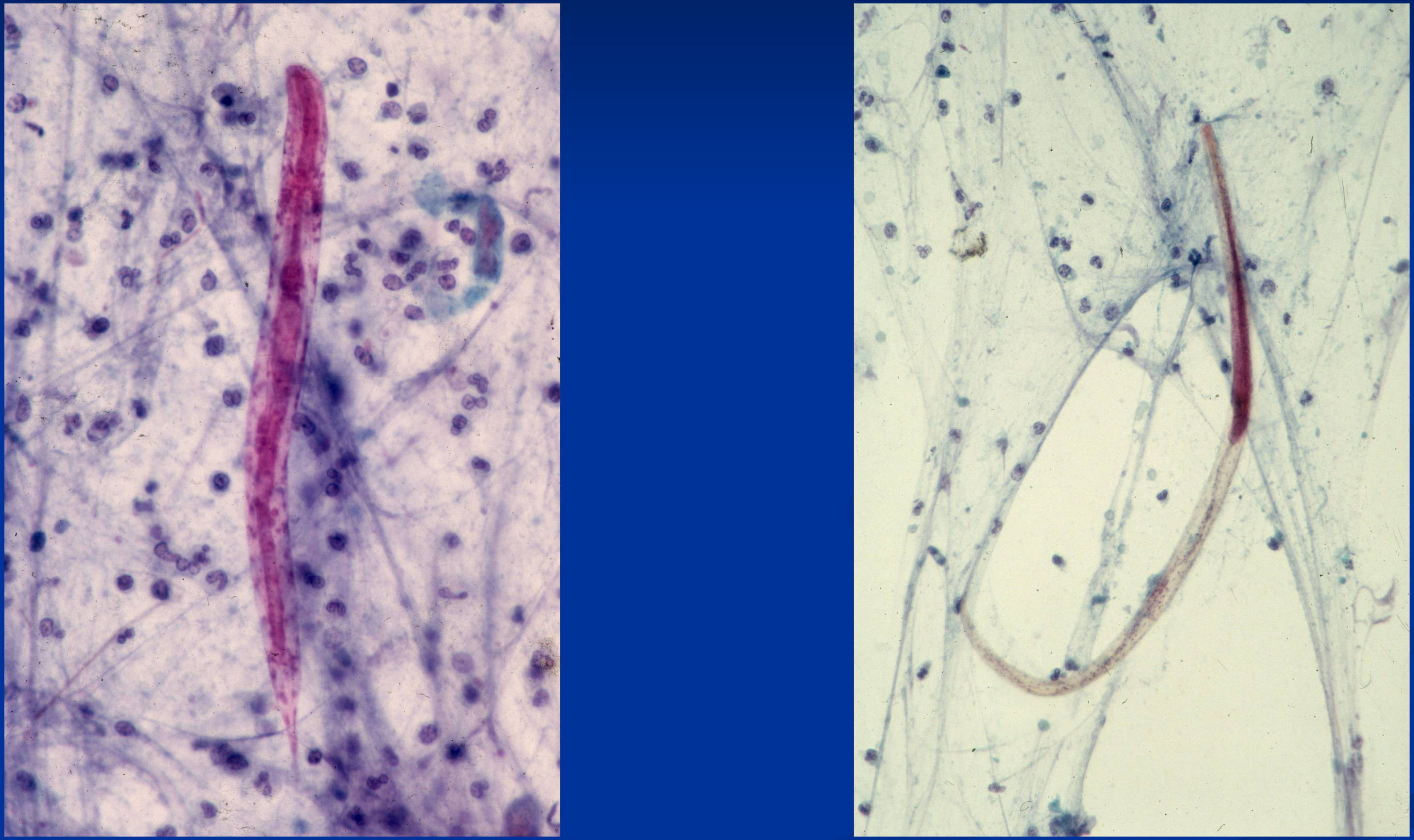
- PP 3-4 wks
- Sensitization from repeated infection, pruritis, erythema
- Larva currens:
  - From unsuccessful migration to lungs, instead migrate through skin to form rapidly progressing linear lesions with sudden onset, a zone of progression and raised red tender lesions
  - Lasts 12-48 hours
- Focal erythema, edema, urticaria, petechiae

Pulmonary Phase

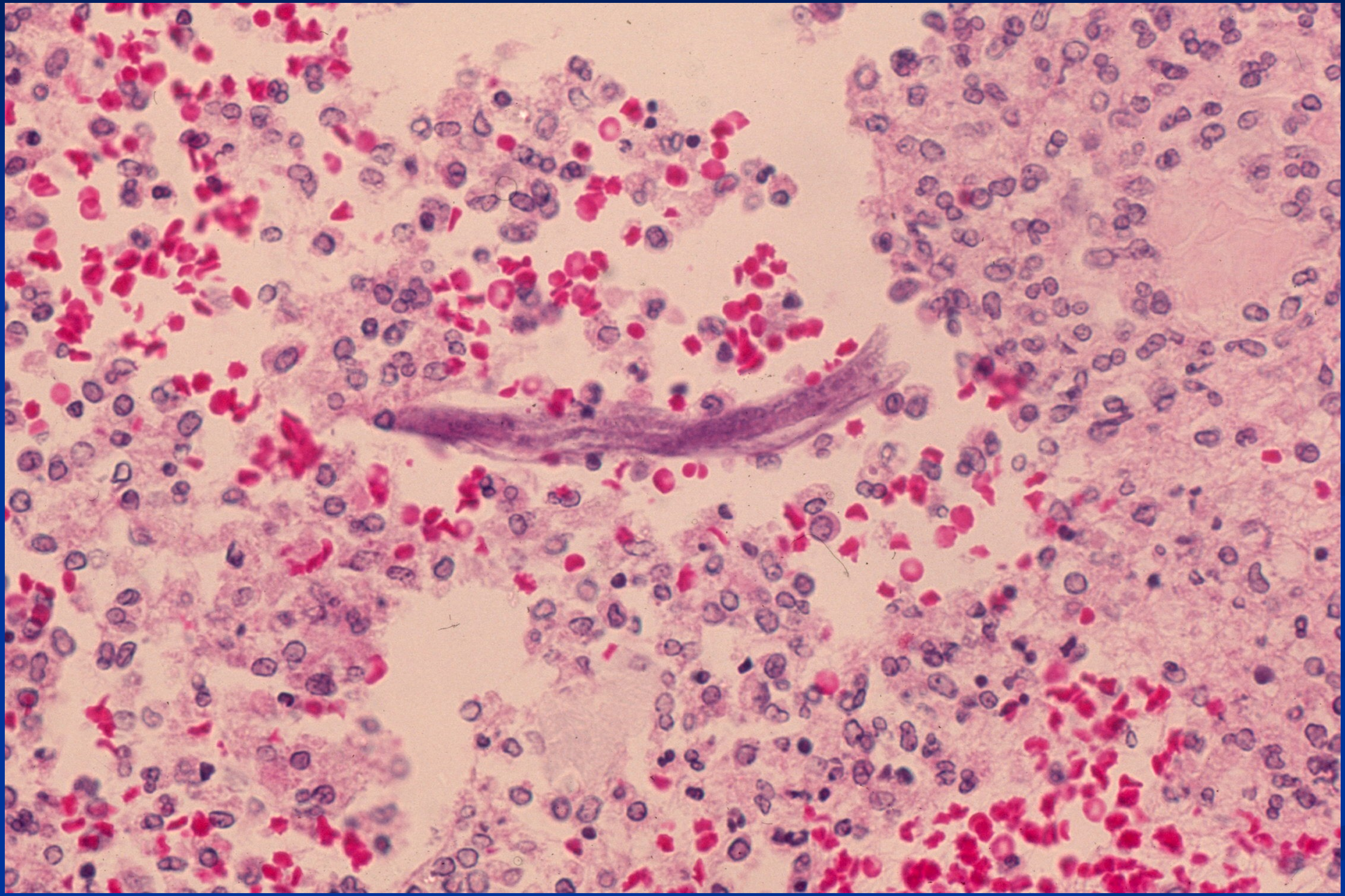
- Larvae breaking into alveoli cause hemorrhage, cellular infiltrates
- Pneumonia-like rxns from sensitization
- Larvae may mature to adulthood in lungs to produce bronchitic or asthmatic symptoms w/ eggs & larvae in sputum
- Throat irritation, dry cough



Strongyloides L1 & L3 in sputum



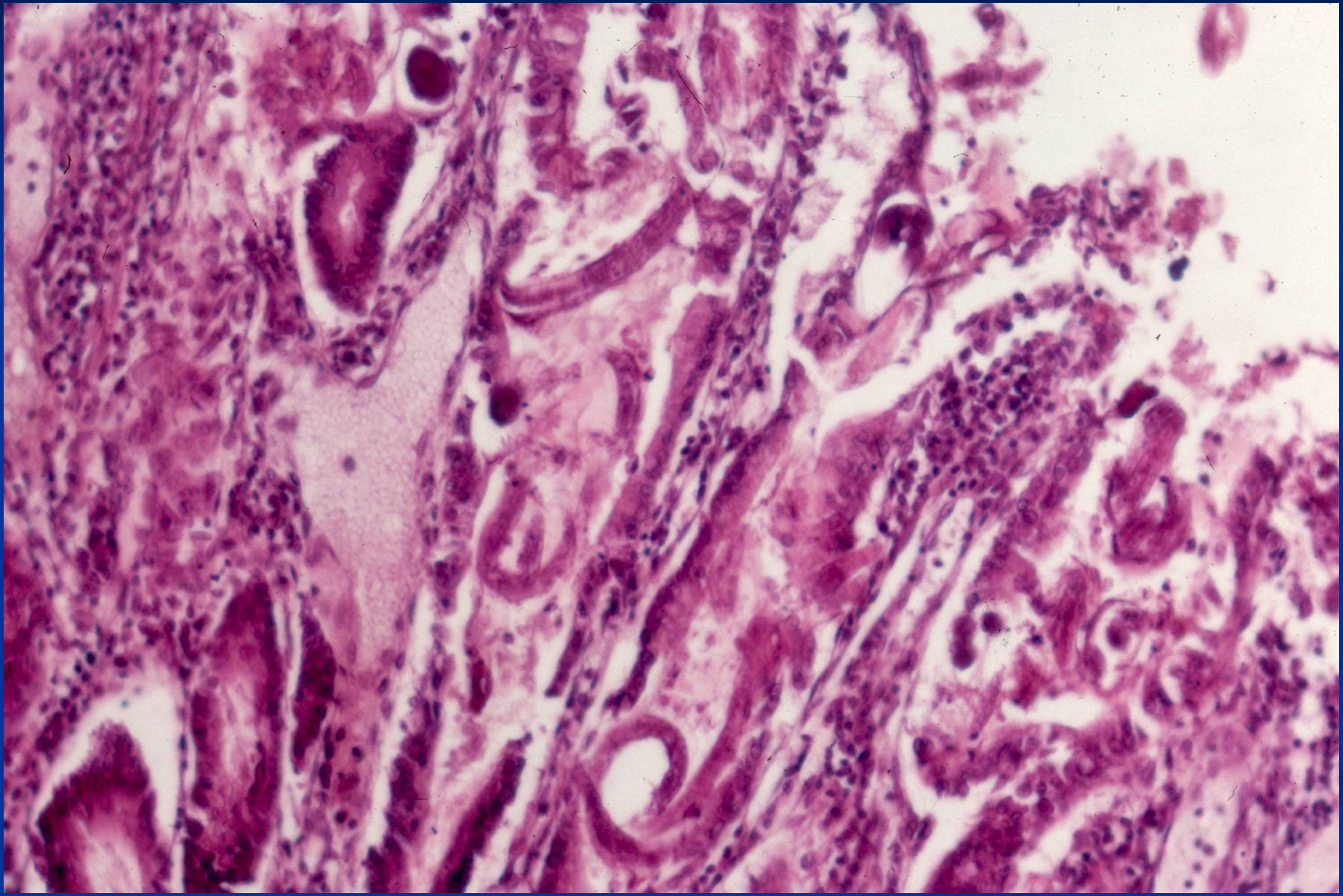
Strongyloides larvae in lung



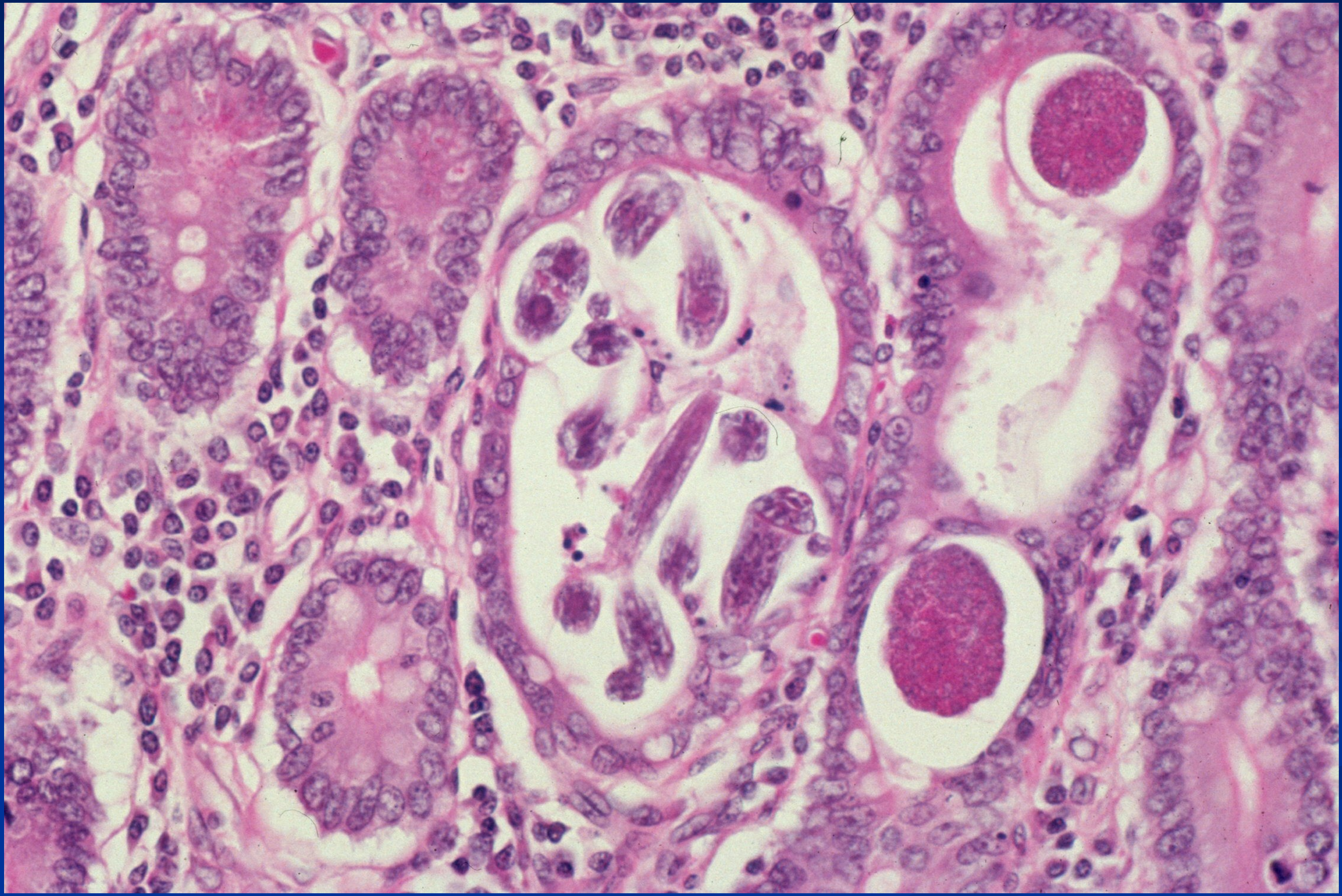
Intestinal phase

- Mucosa contains all stages – adults, larvae, eggs
- Inflammatory response w/ eosinophilic & lymphocytic infiltrates, mucosal atrophy, fibrotic changes, ulcerative colitis
- Peripheral eosinophilia of 50-75%
- Resembles peptic ulcer
- Diffuse abdominal pain, diarrhea
- Can last for years

Strongyloides in intestine



Strongyloides in intestine



Hyperinfection

- Immunosuppresion
- Peripheral eosinophilia absent
- All stages at all gut levels
- Larvae can disseminate to all tissues
- Bowel perforation, meningitis, brain infarction & abscess, respiratory failure, septicemia

Diagnosis

- Larvae in stool (must be differentiated from hookworms)
- Harada-Mori filter paper or charcoal culture of stool
- High eosinophilia during larval migration
- IFA & ELISA available

Treatment

- Due to long term nature of disease, autoinfection & hyperinfection, all persons w/ strongyloidiasis should be treated
- Ivermectin 200 µg/kg/d x 2d
- Albendazole 400 mg po qd x 3d
- Thiabendazole 25 mg/kg po bid x 2d or 7-10d for hyperinfection
- Seropositive individuals undergoing immunosuppressive therapy should possibly by treated for Strongy before suppression

Trichinosis

Trichinosis Epidemiology

- Worldwide where infected meat is consumed.
- Acquired from ingesting encysted first stage larvae in raw or poorly cooked pork, wild boar, bear, walrus, meat of other carnivores, horsemeat
- Garbage feeding of pigs
- Rats can serve as reservoir
- Outbreaks in US associated w/ home-reared pork, wild game, ‘country sausage’

Epidemiology (cont)

- Wild animal meat increasing in importance over domestic pork as human source
- Bear meat caused 38% of cases in US in ’86
- In former USSR, most cases associated with bear & wild boar
- In Africa, from bush pigs & wart hogs
- C & S America, pork is usual source
- Horsemeat in France & Italy (<2000 cases since early 1980’s)
- Cases in US
  - 1990 – 129   1994 – 35   1998 - 19

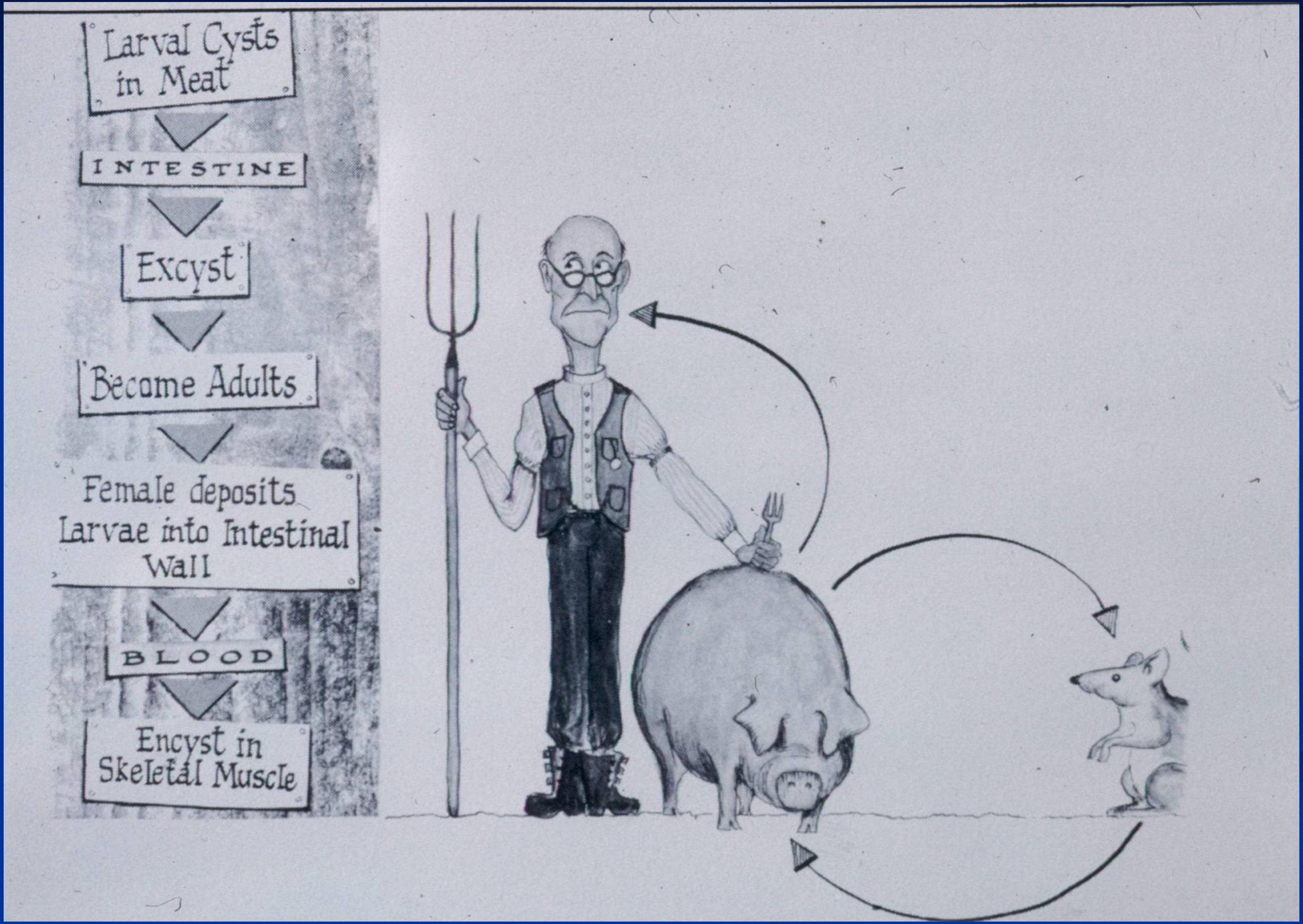
Species in human disease

- *Trichinella spiralis* is usual culprit
- *T. s. nelsoni* in bushpig, warthog, African carnivores
- *T. s. nativa* in Artic carnivores, resistant to freezing

Trichinosis Life cycle

- Larvae encysted in meat released in stomach, migrate to small intestine, penetrate to subepithelium
- Molt 4 times, mature, return to lumen, mate, return to mucosa & produce larvae
- Larvae penetrate mucosa, enter bloodstream, filter out in striated muscle, encyst & wait
- Larvae survive years, eventually die & calcify

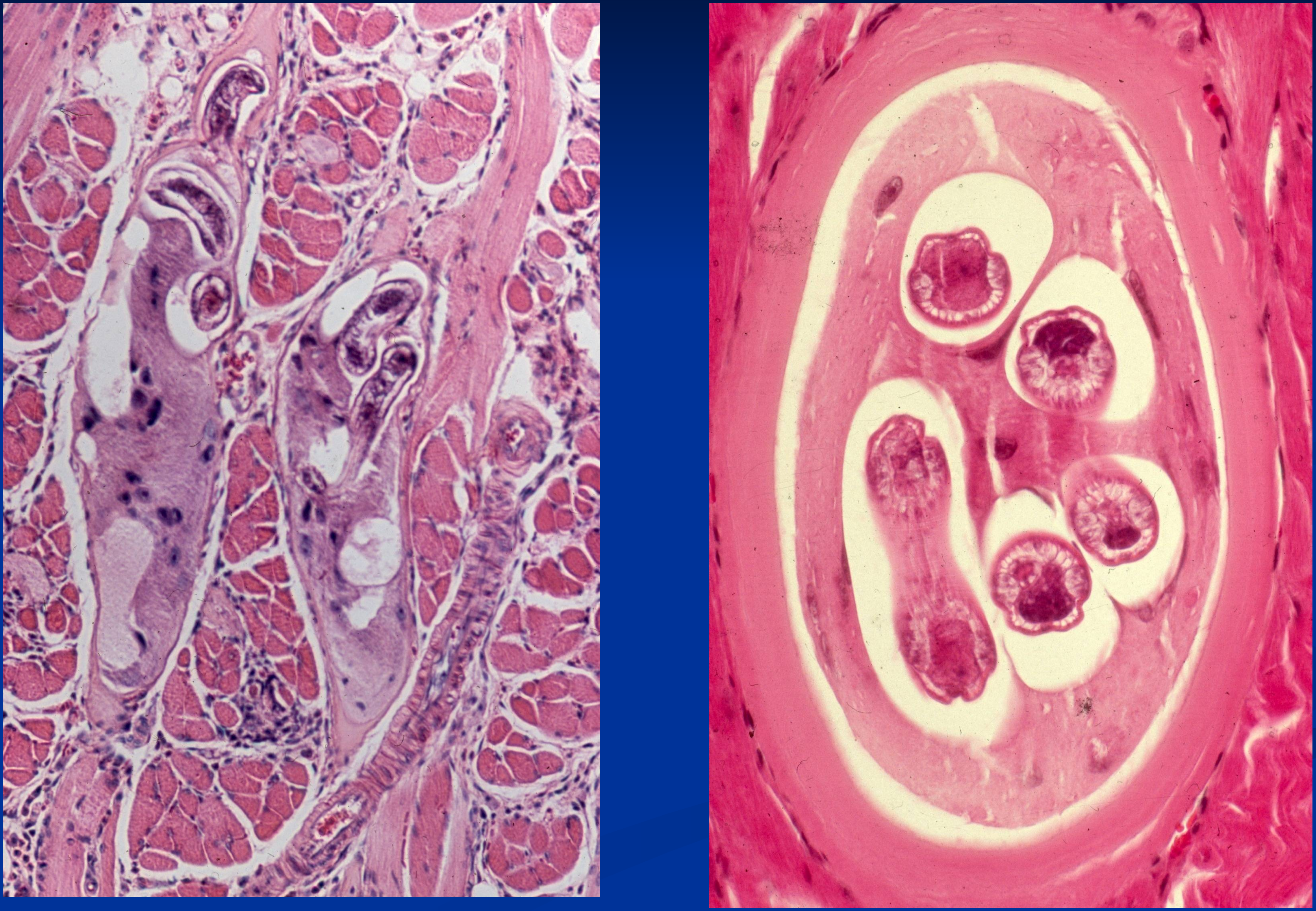
Trichinella life cycle


Larval Cysts in Meat
INTESTINE
Excyst
Become Adults
Female deposits Larvae into Intestinal Wall
BLOOD
Encyst in Skeletal Muscle

Trichinosis Morphology

- Adults
  - Females – 2-3.5 mm x 90 µm, males half that size
  - Larviparous (female produces larvae, not eggs)
- Larvae
  - 1 mm
  - Coiled & encysted in “nurse cell”, an altered muscle cell
  - Cysts lie parallel to fibers
  - 0.4 x 0.26 mm

Encapsulated larva



Trichinosis Intestinal Phase

- Pathology
  - Edema, hyperemia, increased mucous production, eosinophilic infiltrates

- Clinical
  - Occurs within first week after ingestion
  - Nausea, cramps, vomiting, mild fever, appetite loss, mild diarrhea or constipation, headache, weakness

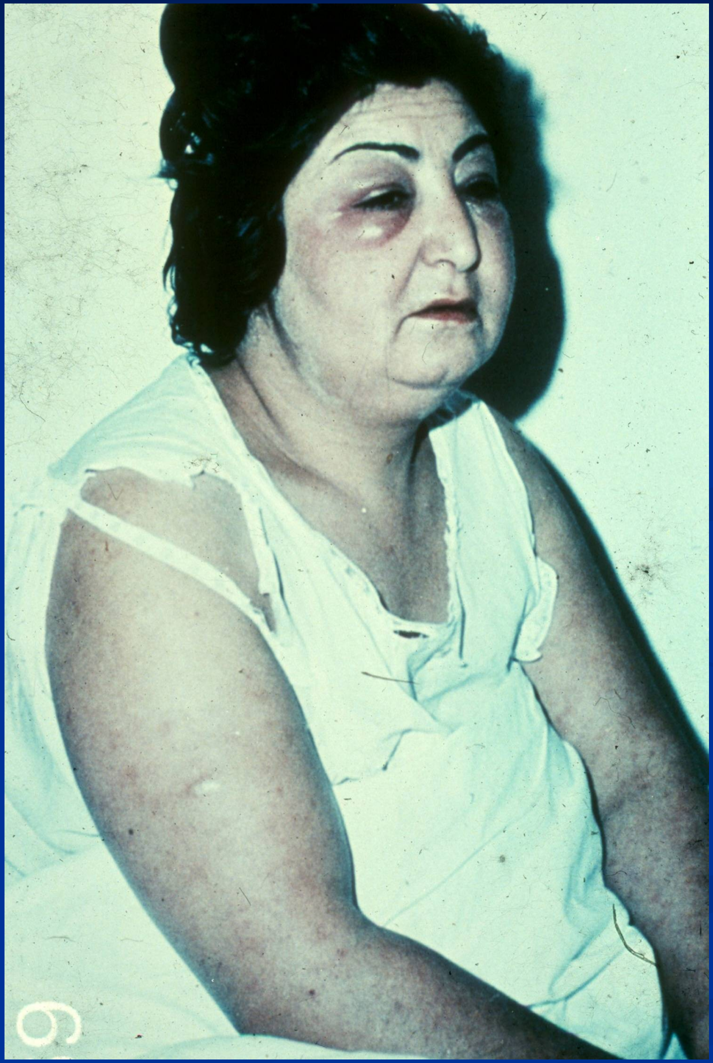
Trichinosis Muscle Phase

- Usually affected are diaphragm, tongue, masseter, intercostal, extraocular, and laryngeal muscles
- Edema, hyperemia, lymphocytic & eosinophilic infiltrates, striation loss
- Occurs 2nd week after ingestion
- Fever, periorbital edema, myalgia, weakness, headache, rash, pruritis, dry cough

Muscle Phase (cont)

- Splinter hemorrhages under fingernails
- Eosinophilia up to 90% by 3-4 wks
- Myocarditis frequent complication
- Recovery in 2-6 months
- 2% US cases fatal, usually from massive infection

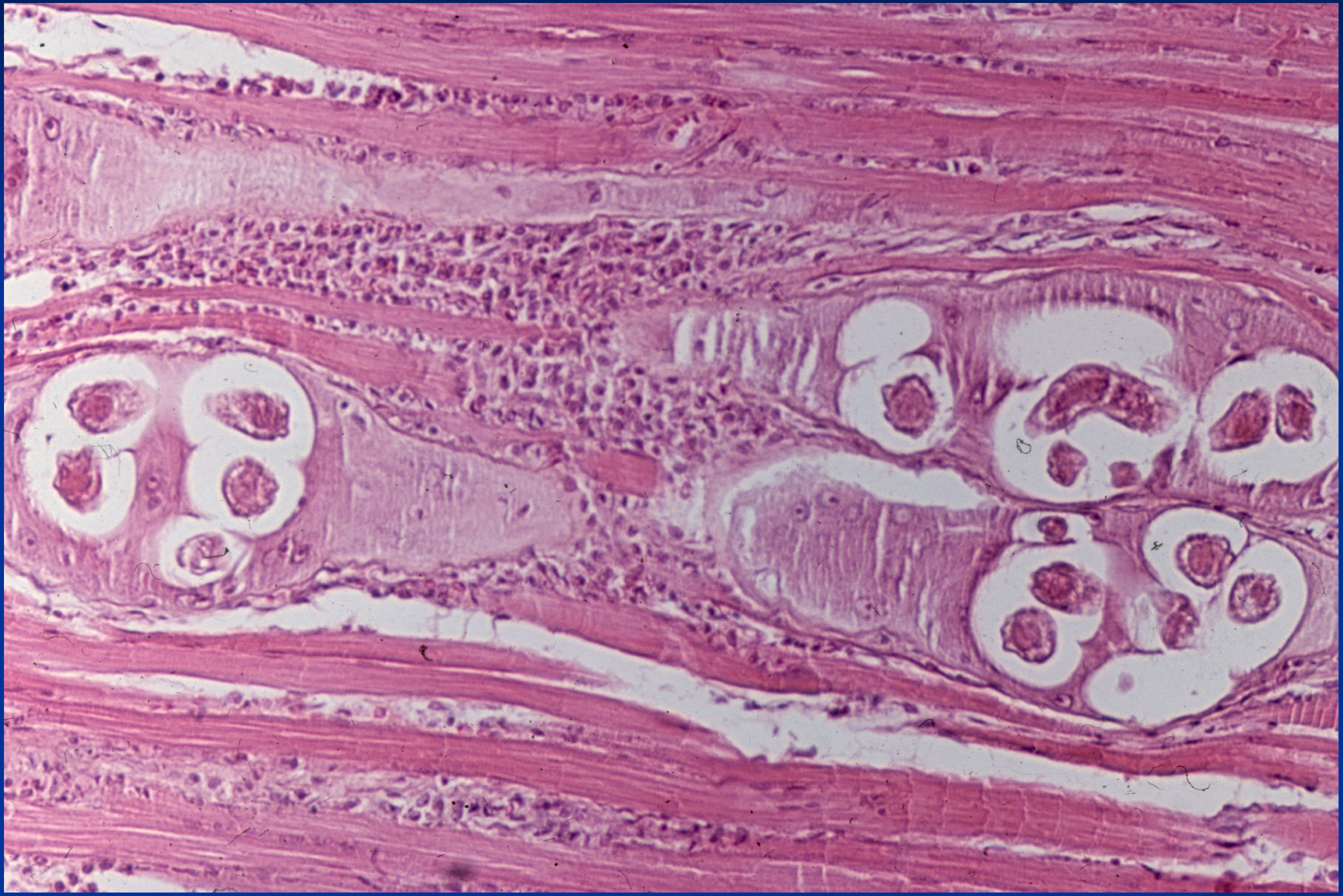
Periorbital edema



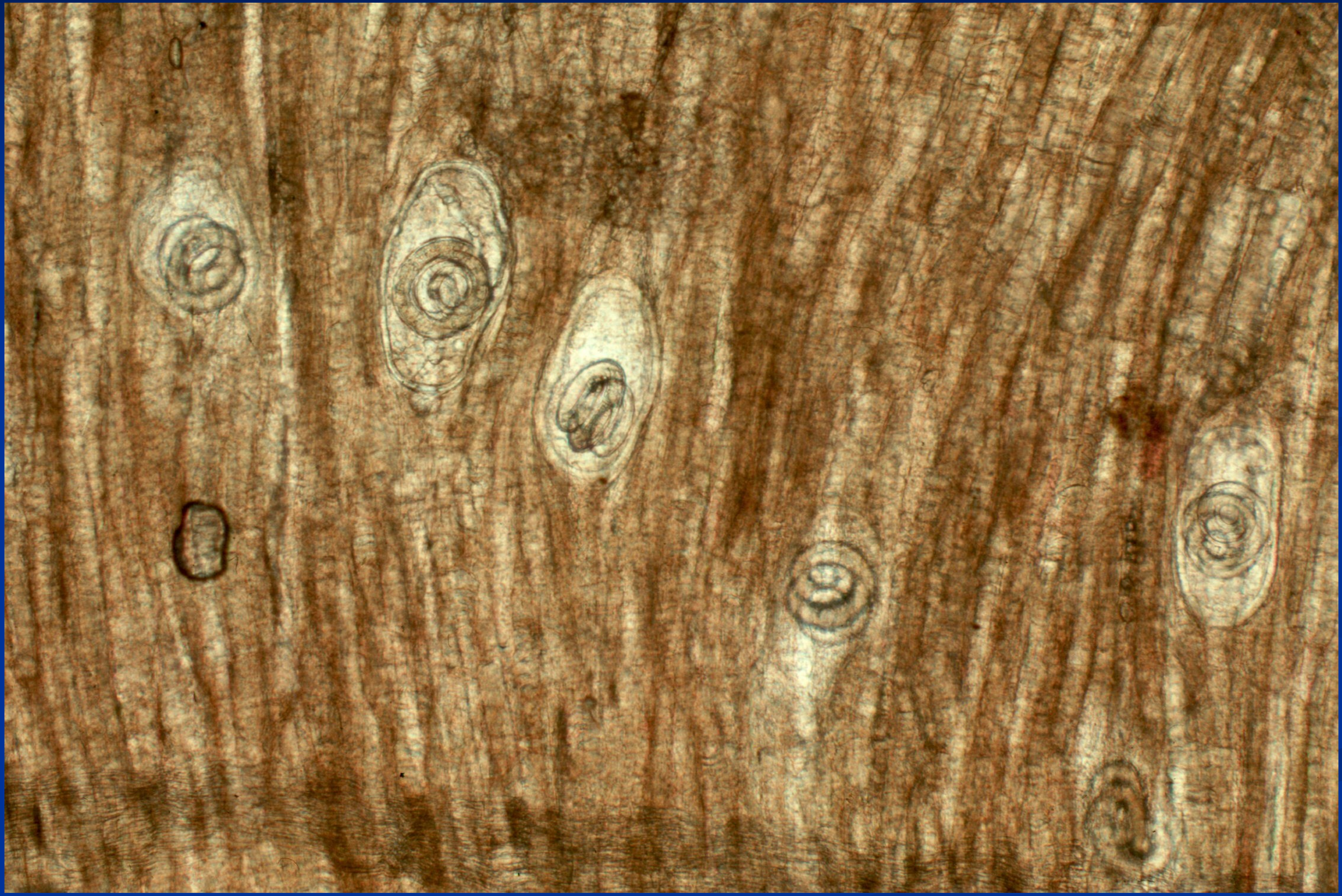
Trichinosis Diagnosis

- Encapsulated larva seen in superficial muscle biopsy
  - 4-6 wks post-infection gives highest yield.
  - Part of specimen histologically prepared
  - Rest is digested in 1% pepsin 1% HCl and filtrate examined or compressed between glass slides & examined under microscope
- History of exposure

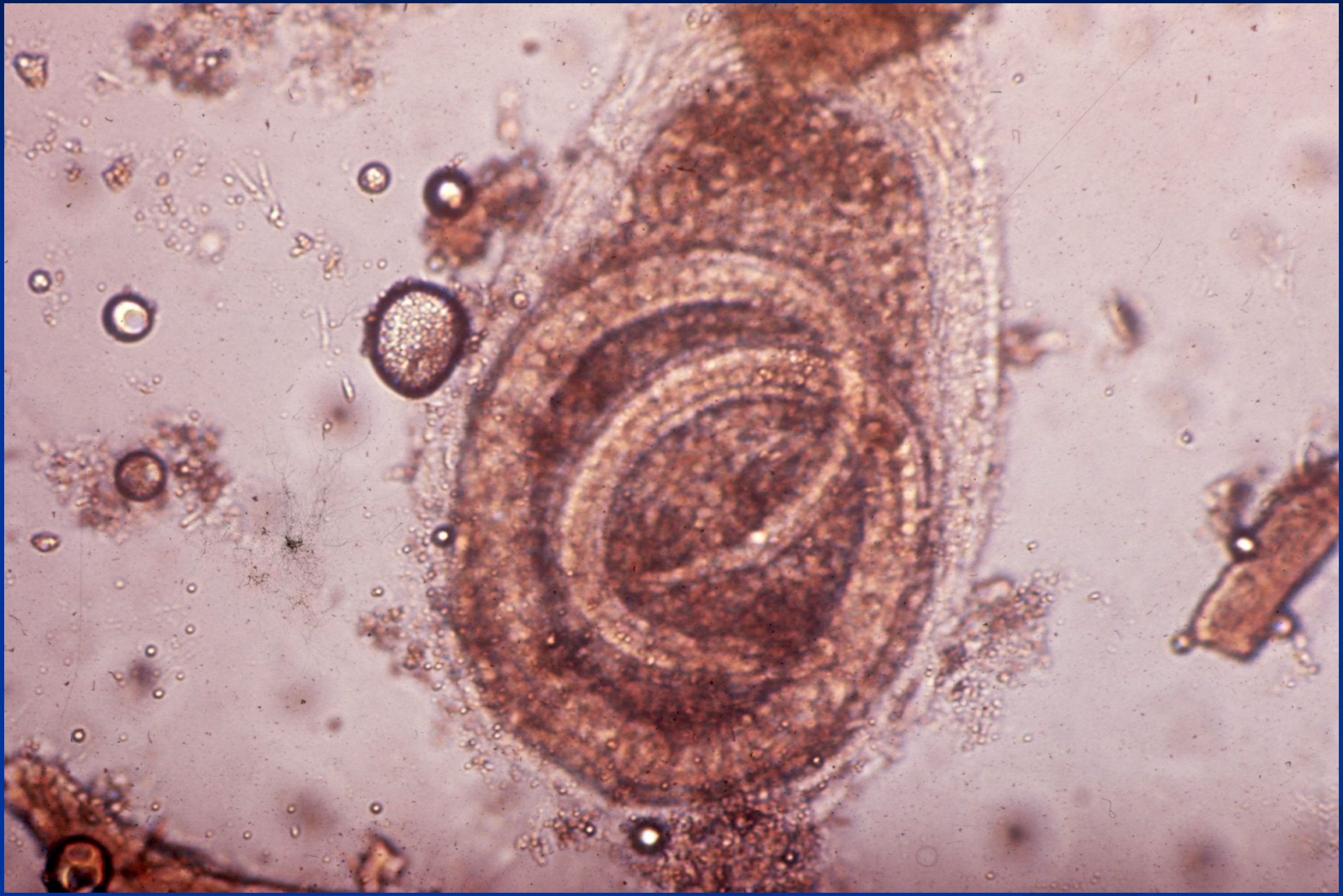
Encysted larvae in diaphragm (10x)



Encysted larvae in squash prep of diaphragm (4x)



Larva from pepsin digest of diaphragm



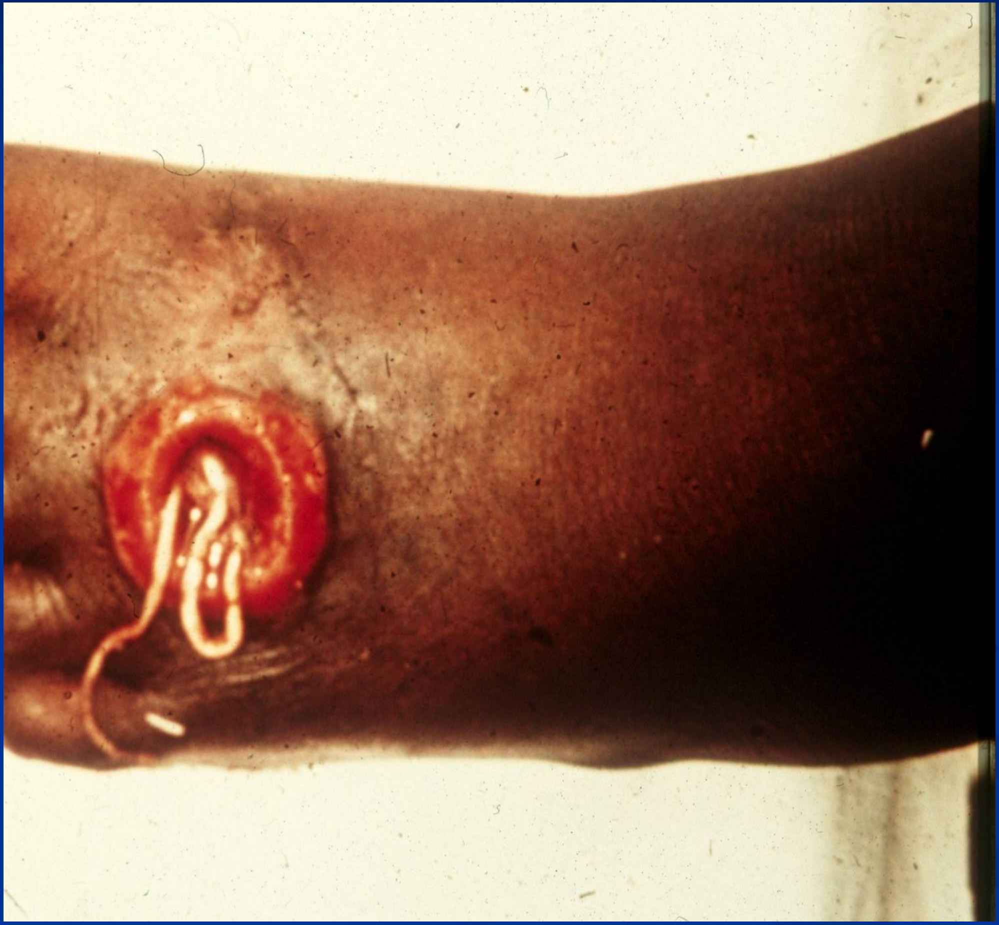
Trichinosis Dx & Tx

- Bentonite Flocculation Test (BFT)
  - 4-fold rise or 1:5 titer considered positive
  - + at 3 wks, falls after 1-2 years
- ELISA, IFA
- Corticosteroids for inflammation
- Albendazole 400 mg po bid x14d
- Mebendazole 5 mg/kg bid x10-13d

Prevention

- Thorough cooking of suspected meat
- Freezing, except for walrus and bear meat infected with *T. nativa*
- Pre-cooking of offal & garbage before feeding to pigs
- Strict regulation of commercial production

Dracunculus medinensis
The Guinea Worm



Dracunculiasis Epidemiology

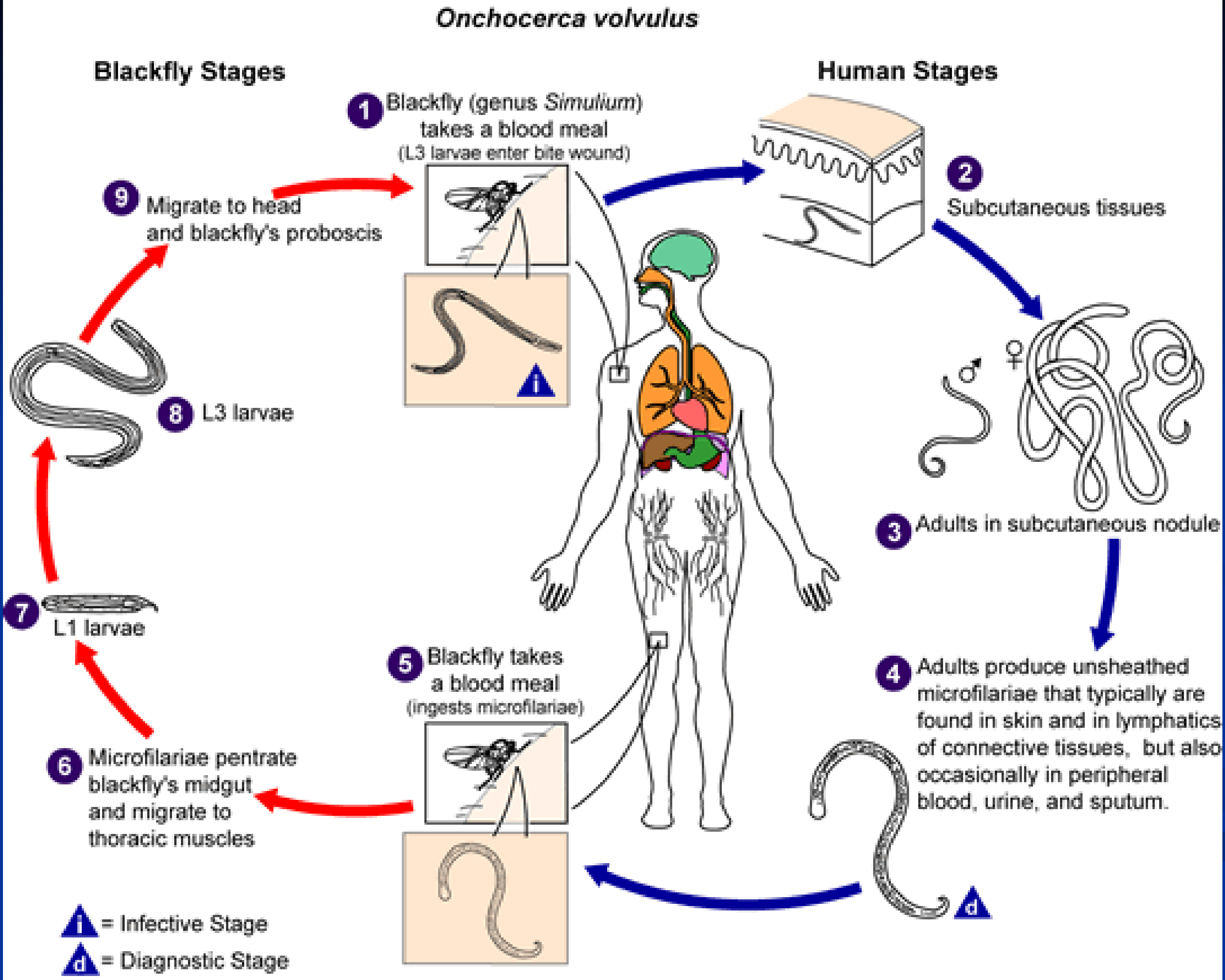
- *Dracunculus medinensis*, Guinea worm, fiery serpent
- India, W&C Africa, Middle East.
- 3.3 million cases in 1986; 75k in 2000 due to Eradication Initiative
- Economic loss is from morbidity, rarely fatal
- Acquired by drinking the infective third stage larvae in copepods, the intermediate host

Epidemiology (cont.)

- Focal distribution associated with water sources (step wells, ponds, water holes)

Dracunculiasis Life Cycle

- Females in human host extrude uterus from blister into water & release larvae (1-3 million)
- Larvae ingested by copepods and molt to third stage in 12-14 days
- Infected copepods swallowed by human, larvae emerge, penetrate gut wall, migrate via lymphatics & peritoneal tissue to SQ tissue (usually of legs & feet), mature, mate
- Male fizzles out & dies, female produces larvae for 2 wks, then dies



Onchocerca volvulus

Blackfly Stages

Human Stages

1 Blackfly (genus *Simulium*) takes a blood meal (L3 larvae enter bite wound)

2 Subcutaneous tissues

3 Adults in subcutaneous nodule

4 Adults produce unsheathed microfilariae that typically are found in skin and in lymphatics of connective tissues, but also occasionally in peripheral blood, urine, and sputum.

5 Blackfly takes a blood meal (ingests microfilariae)

6 Microfilariae pentrate blackfly's midgut and migrate to thoracic muscles

7 L1 larvae

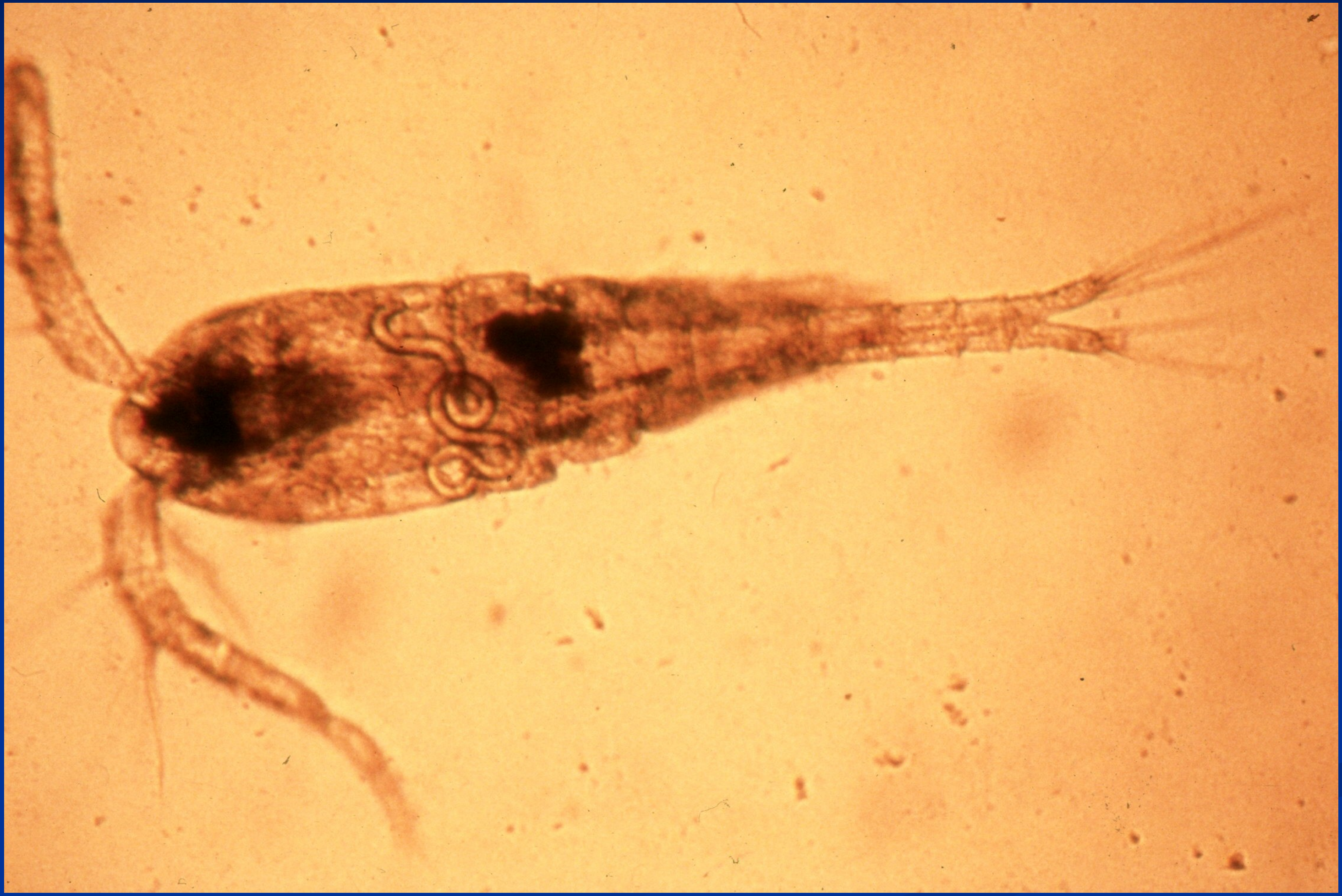
8 L3 larvae

9 Migrate to head and blackfly's proboscis

i = Infective Stage

d = Diagnostic Stage

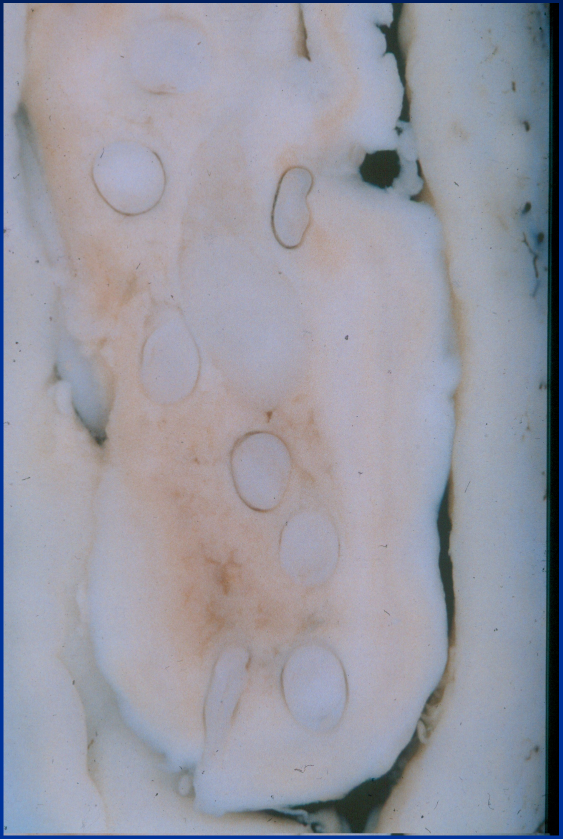
Copepod (*Cyclops sp.)* w/ L3 in thorax



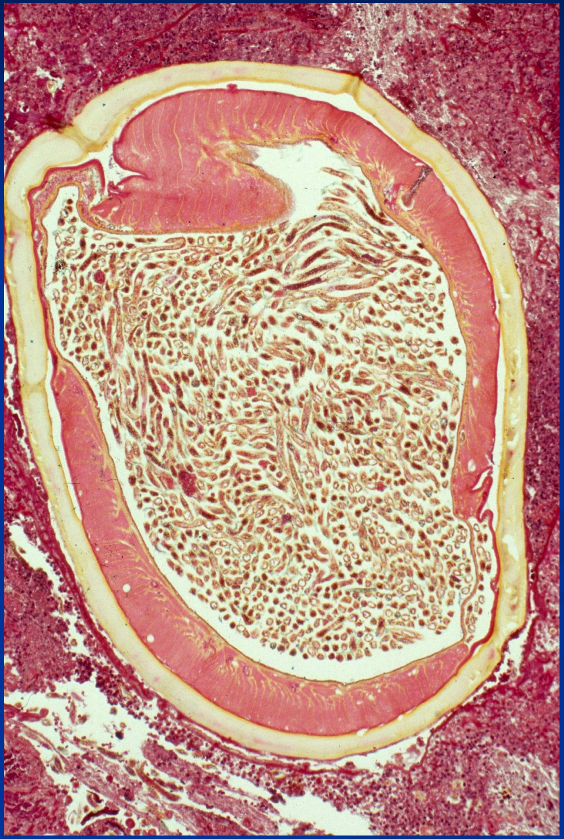
Dracunculiasis Organism

- Adults: females are longest nematode of humans
  - 50-120 cm x 1-2 mm, produces millions of live larvae
  - Males rarely found in tissue, <50 mm
  - In subcutaneous connective tissue
- 1st stage larvae (L1's)
  - 500-750 x 15-25 µm
- 3rd stage larvae (L3's)
  - 240-610 x 12-23 µm

Female *D. medinensis* in eyelid



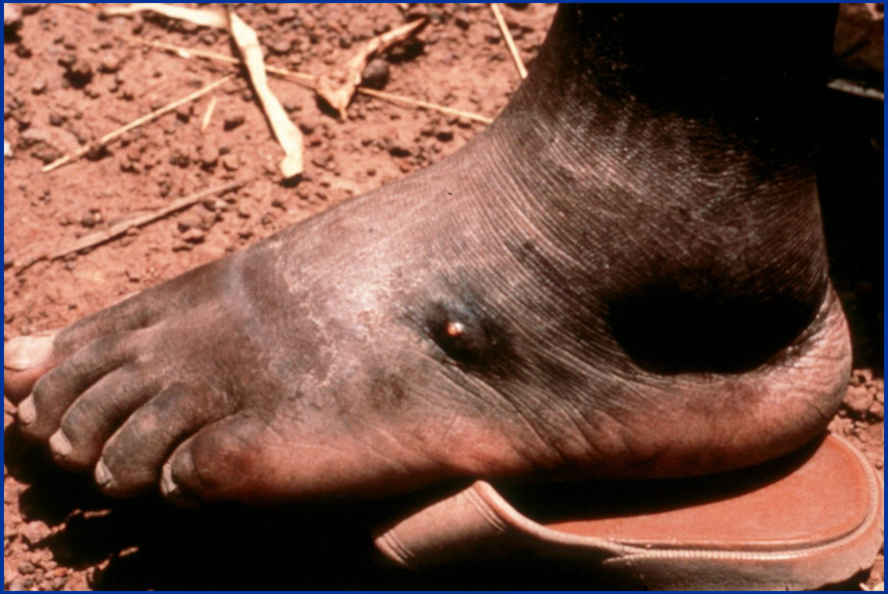
Gross XS, note sections of adult female worm



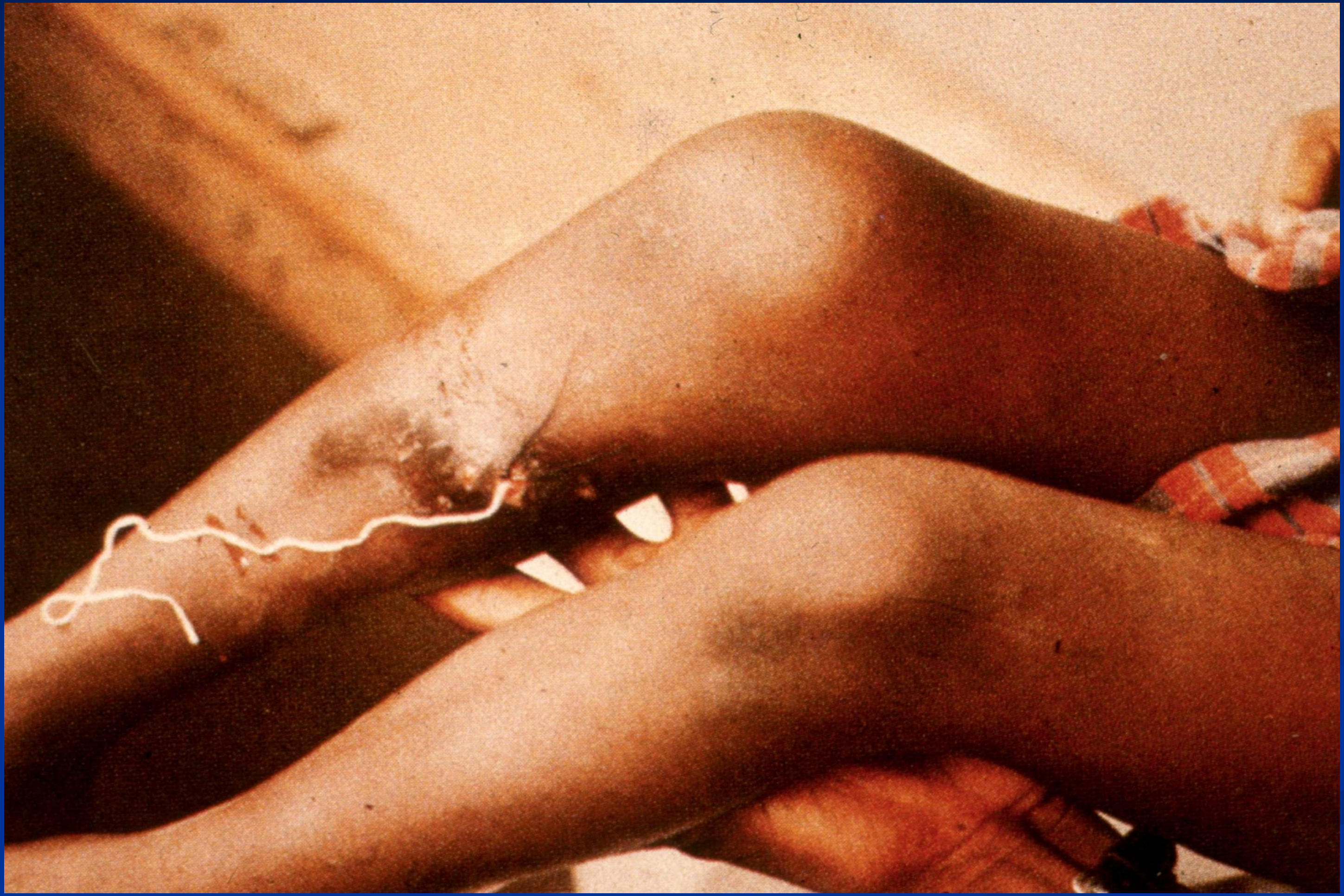
XS of female, note many larvae inside, 2.5x Movat stain

Dracunculiasis Pathology

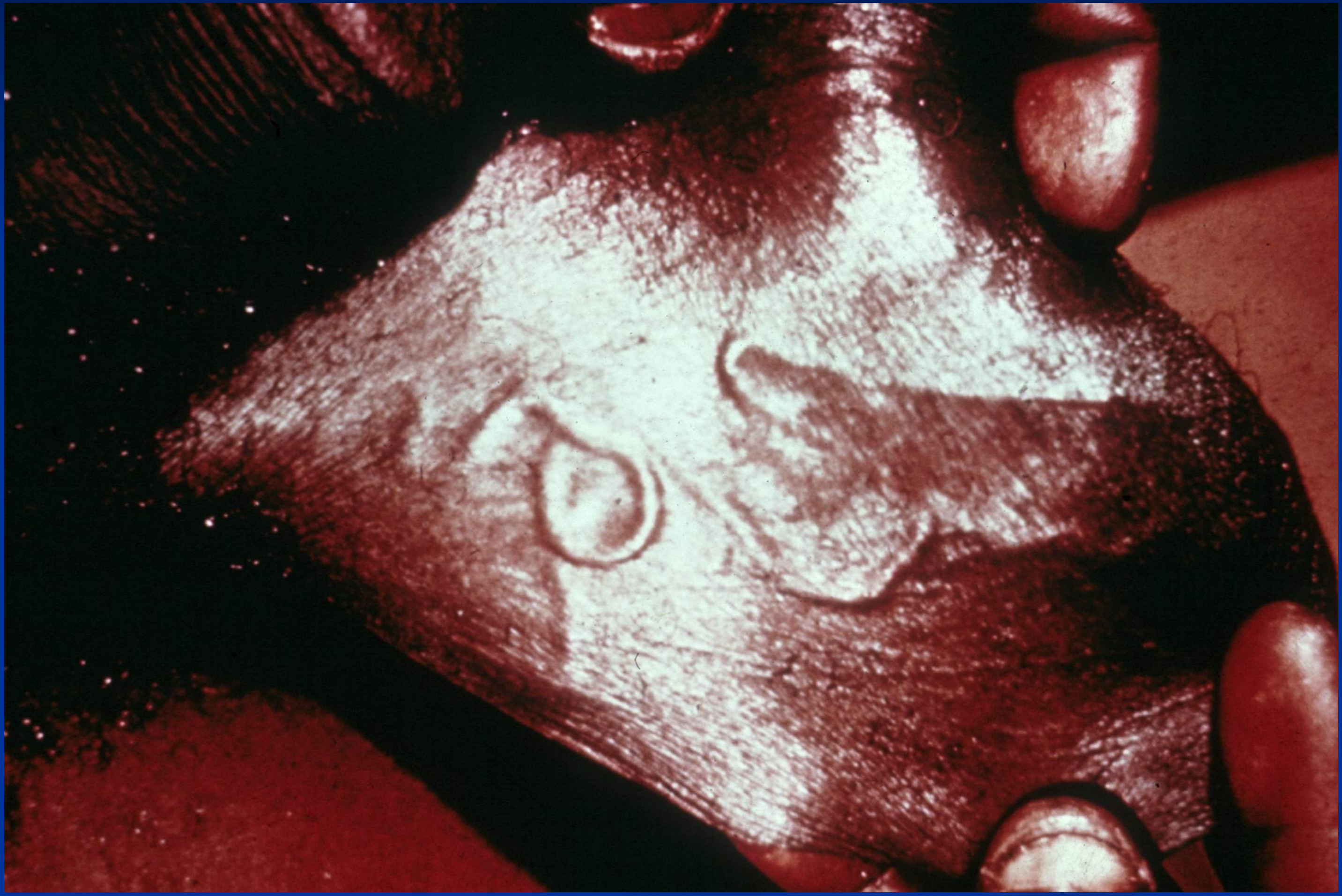
- Blisters from toxic metabolic products
- If female dies, will produce severe inflammatory response w/ eventual fibrosis & calcification
- Can get 2° bacterial infection of ulcer



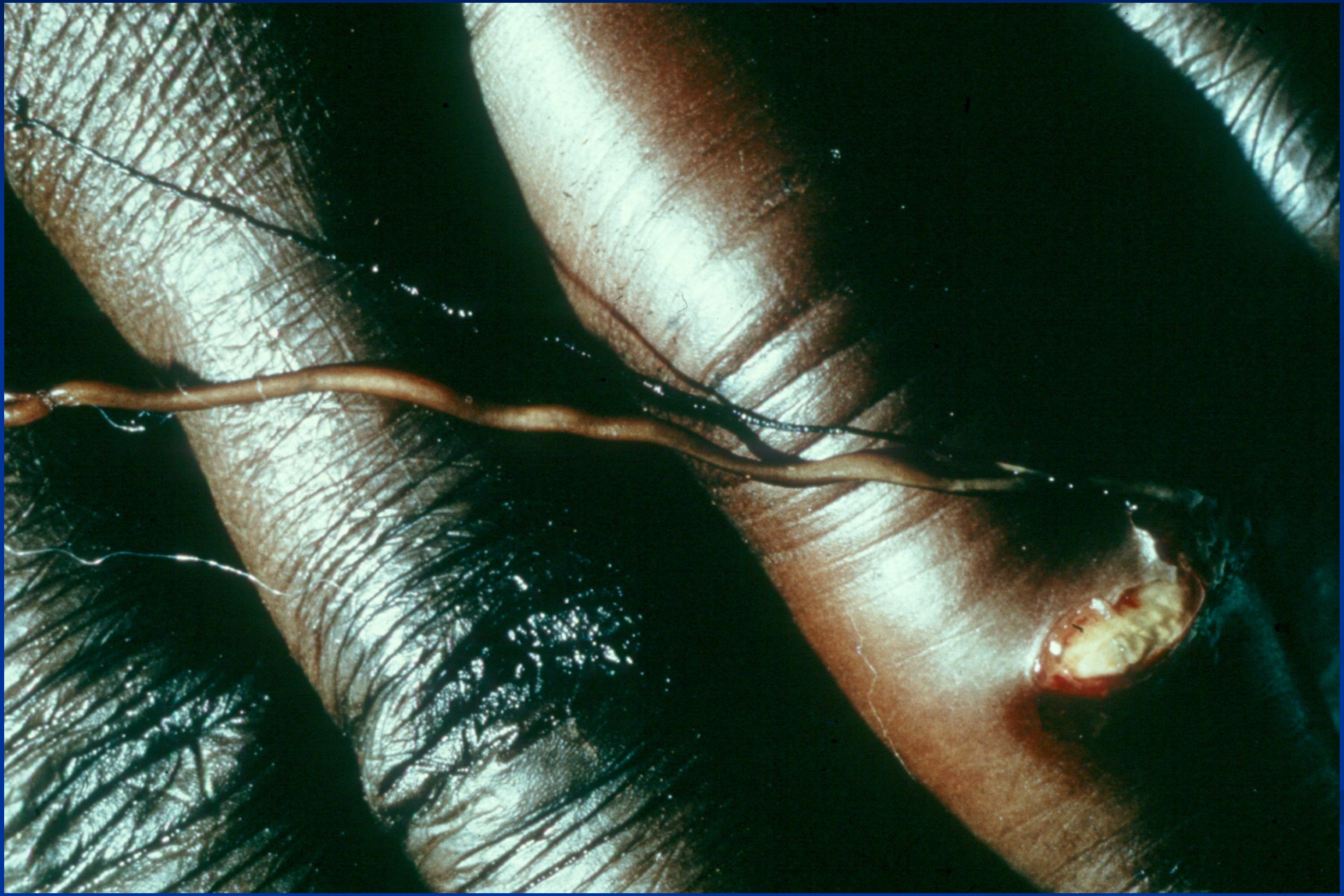
Extruded female in leg



D. medinensis in scrotum



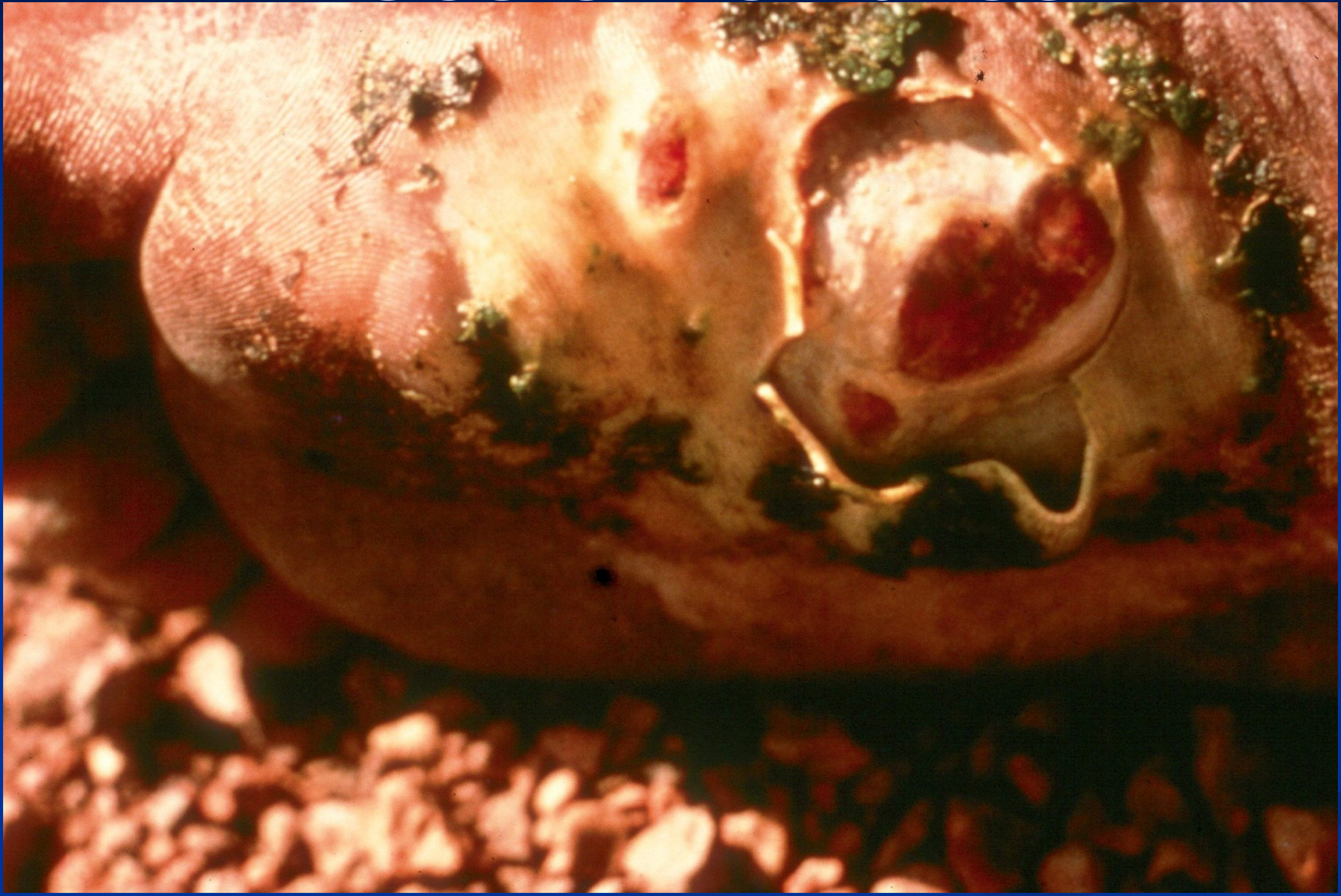
Ulcer & exposed female in hand



Dracunculiasis Clinical

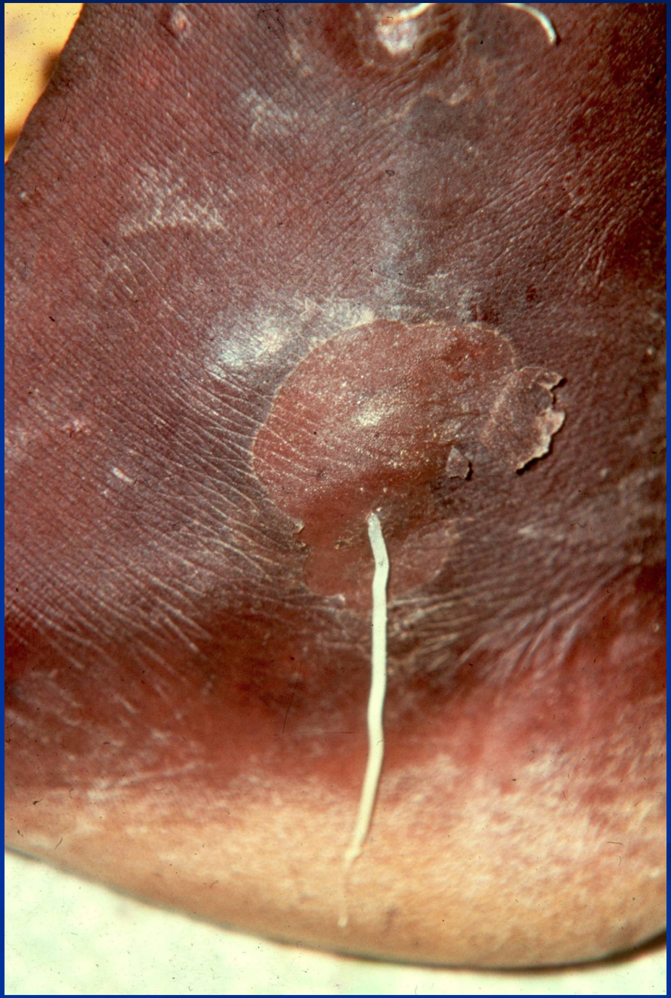
- IP 9-14 months
- May get systemic allergic rxn days before rupture
- Blisters 10-15 mm, pruritic, painful
- Worm emerges over a period of 3-4 wks
- Ulcers a pathway for complications
  - Tetanus, abscess, chronic ulcers, cellulitis, necrosis, gangrene, septicemia, joint problems

Secondary bacterial infection of ulcer



Dracunculiasis Diagnosis

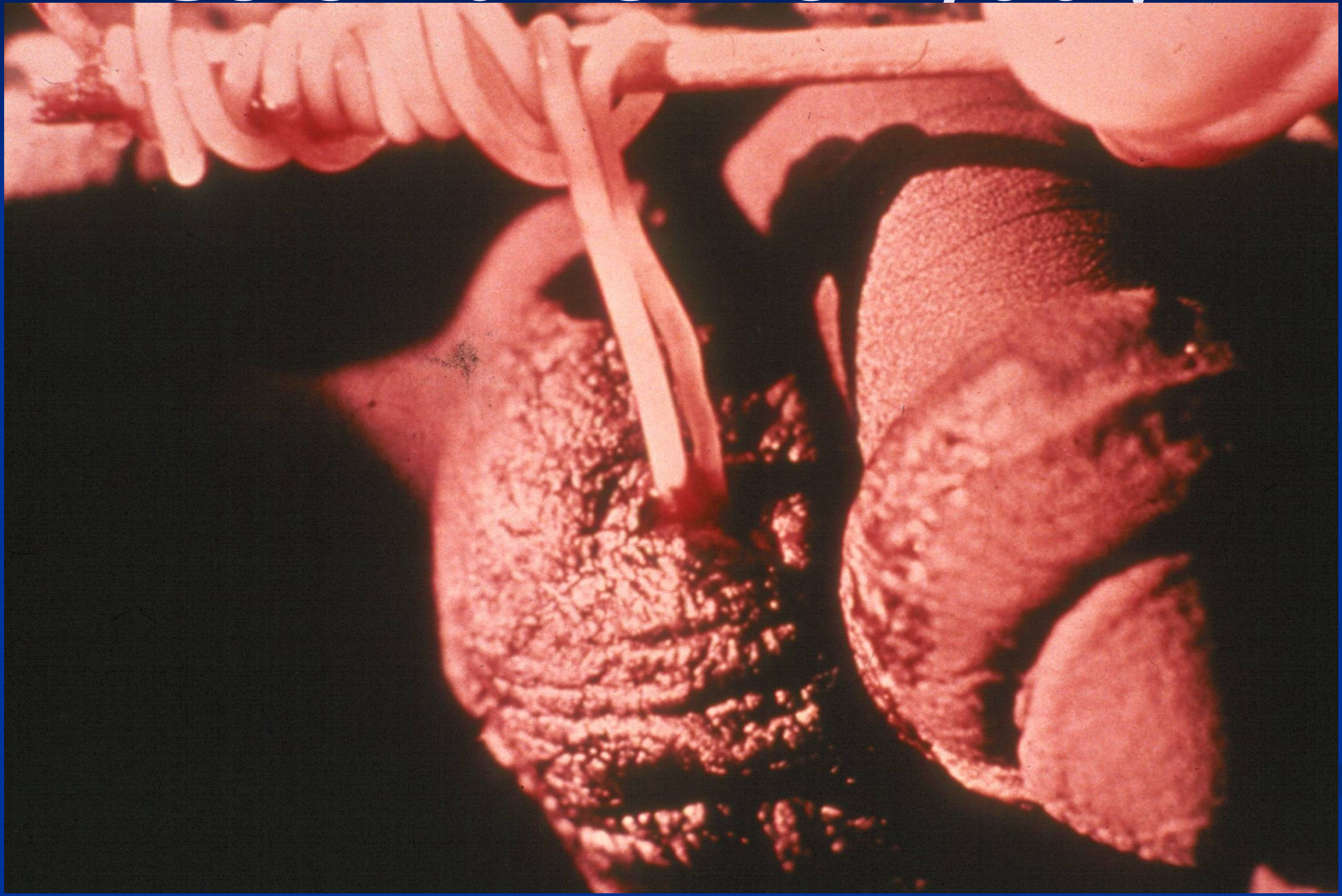
- Blister
- Emergent female
- Calcifications on xray



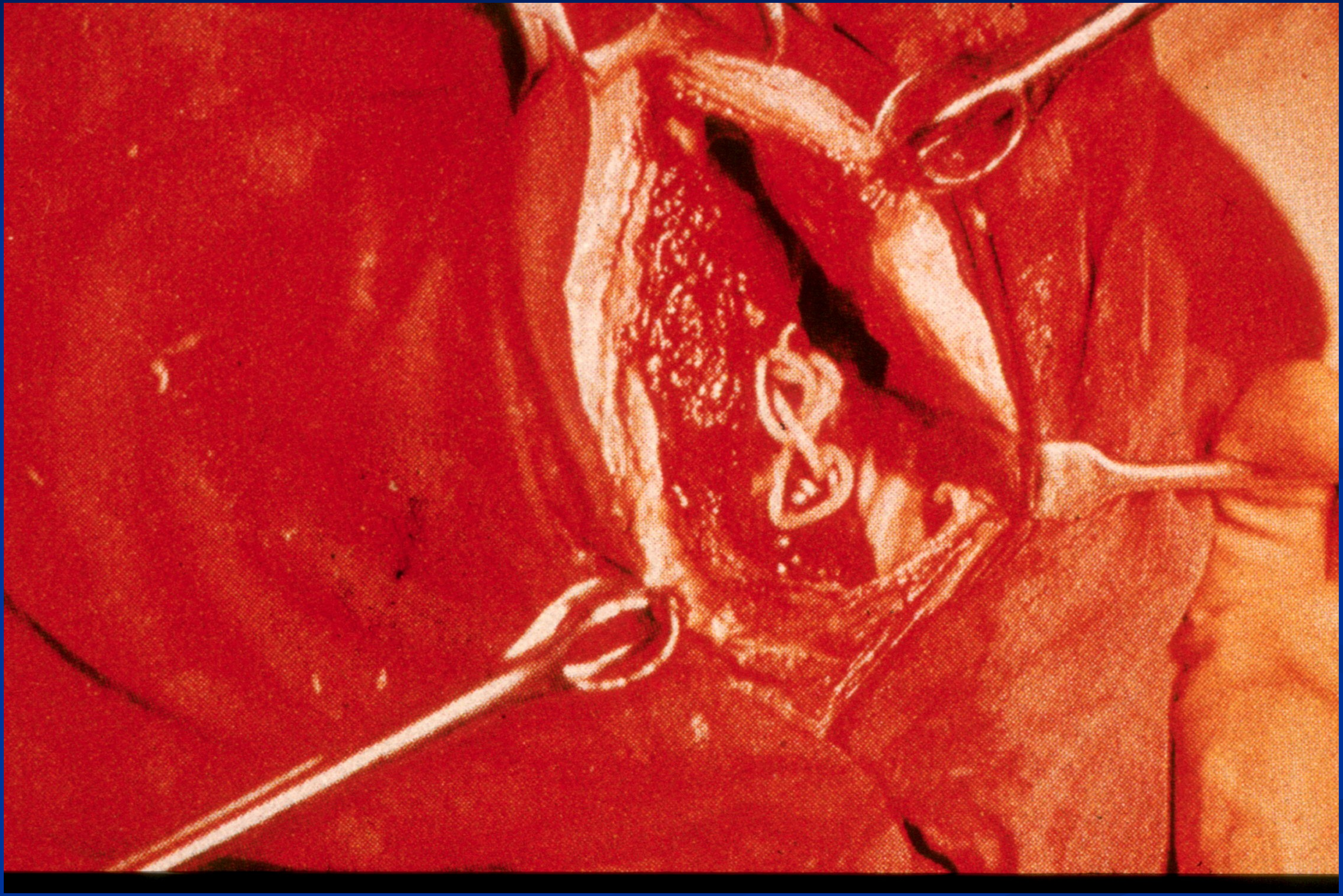
Dracunculiasis Treatment

- Traditional removal - Protruding worm is wrapped around a stick and a few cm are drawn out per day (prevents rupture & anaphylaxis)
- Surgical
- Benzimidazoles not very effective against female but may add in extraction
  - Metronidazole 500 mg po qd to decrease inflammatory response

Traditional removal by wrapping worm around stick a few cm/day



Surgical removal



Dracunculiasis Control

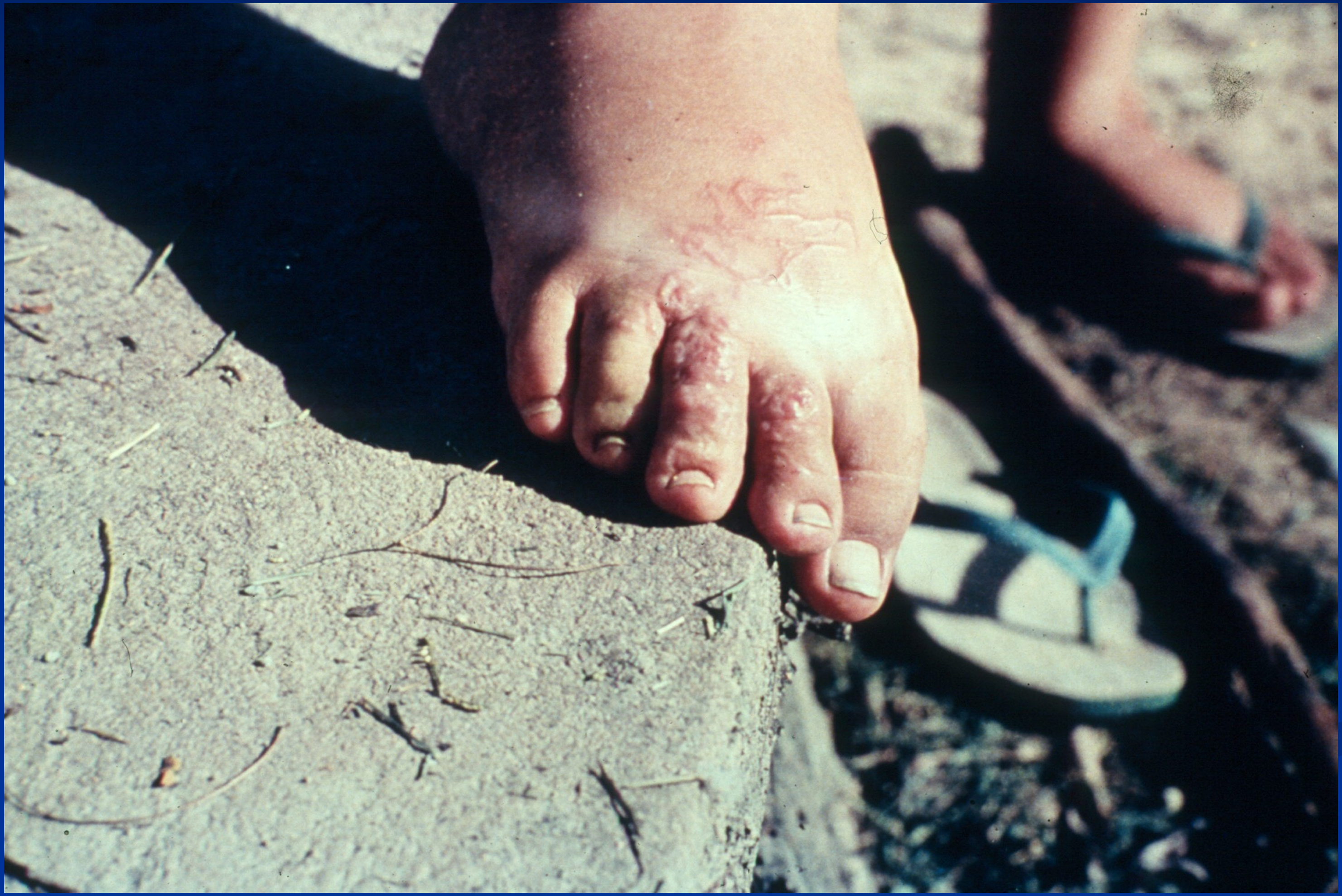
- Minimization of water contact
  - Water pumps
  - Step wells to draw wells, platforms for standing
  - Bandaging of blisters
  - Contain cases, keep from public water sources
- Filtration of drinking water through cloth w/ 120-130 µm mesh
- Public education on prevention
- Treatment of water sources w/ larvicide to kill copepods (temephos)

Intervention

Eradication Initiative

- CDC, Carter Center, UNICEF, WHO, World Bank primary stakeholders
- 98% reduction in number of cases
- 3.3 million cases in 1986; 75k in 2000
- 14 African countries still reported indigenous cases

Larval Migrans Syndromes

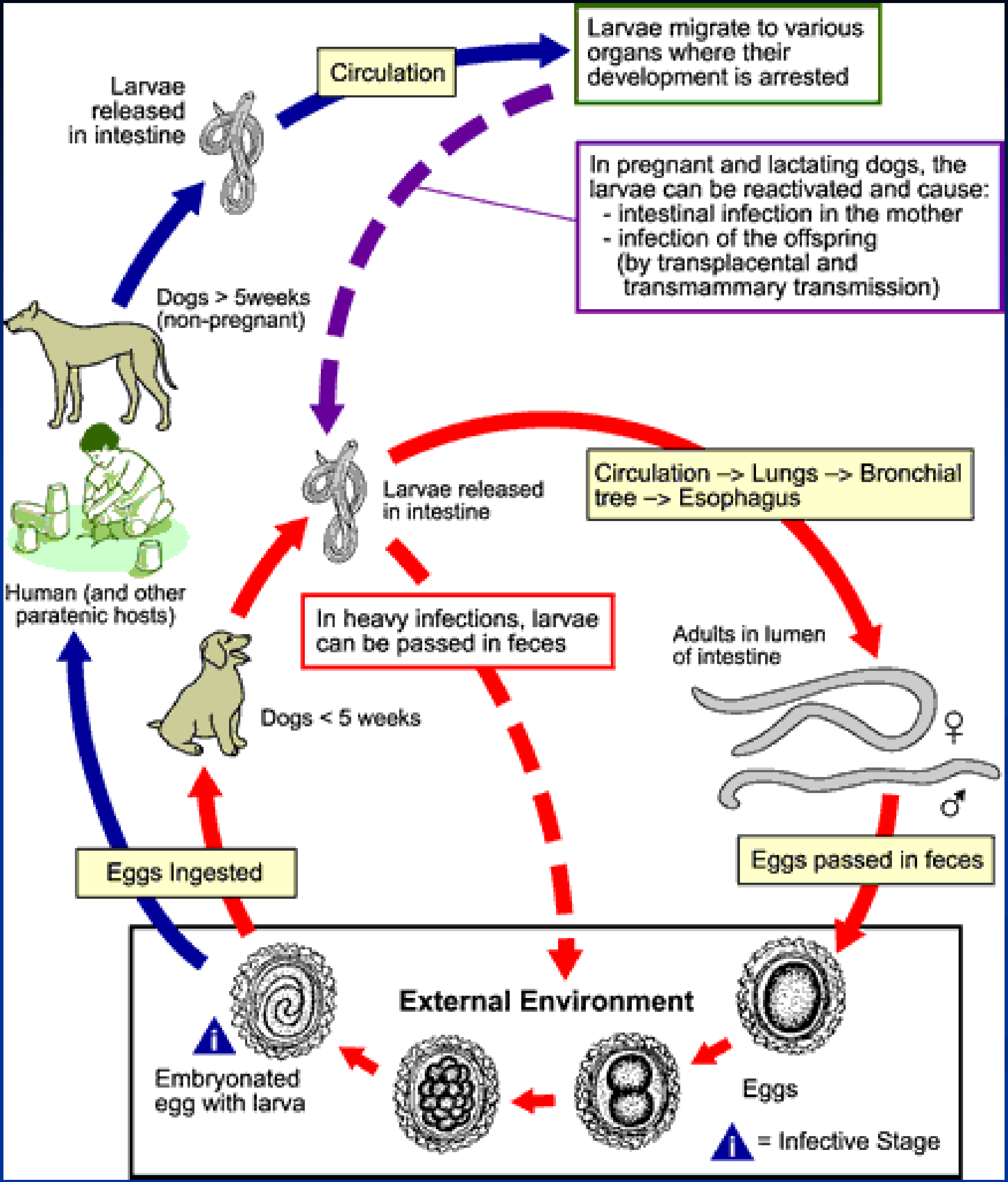


Larval Migrans Syndromes

- Cutaneous larval migrans (CLM)
- Visceral larval migrans (VLM)
- Baylisascariasis
  - Neural (NLM)
  - Ocular (OLM)
- Gnathostomiasis

Cutaneous Larval Migrans (CLM)

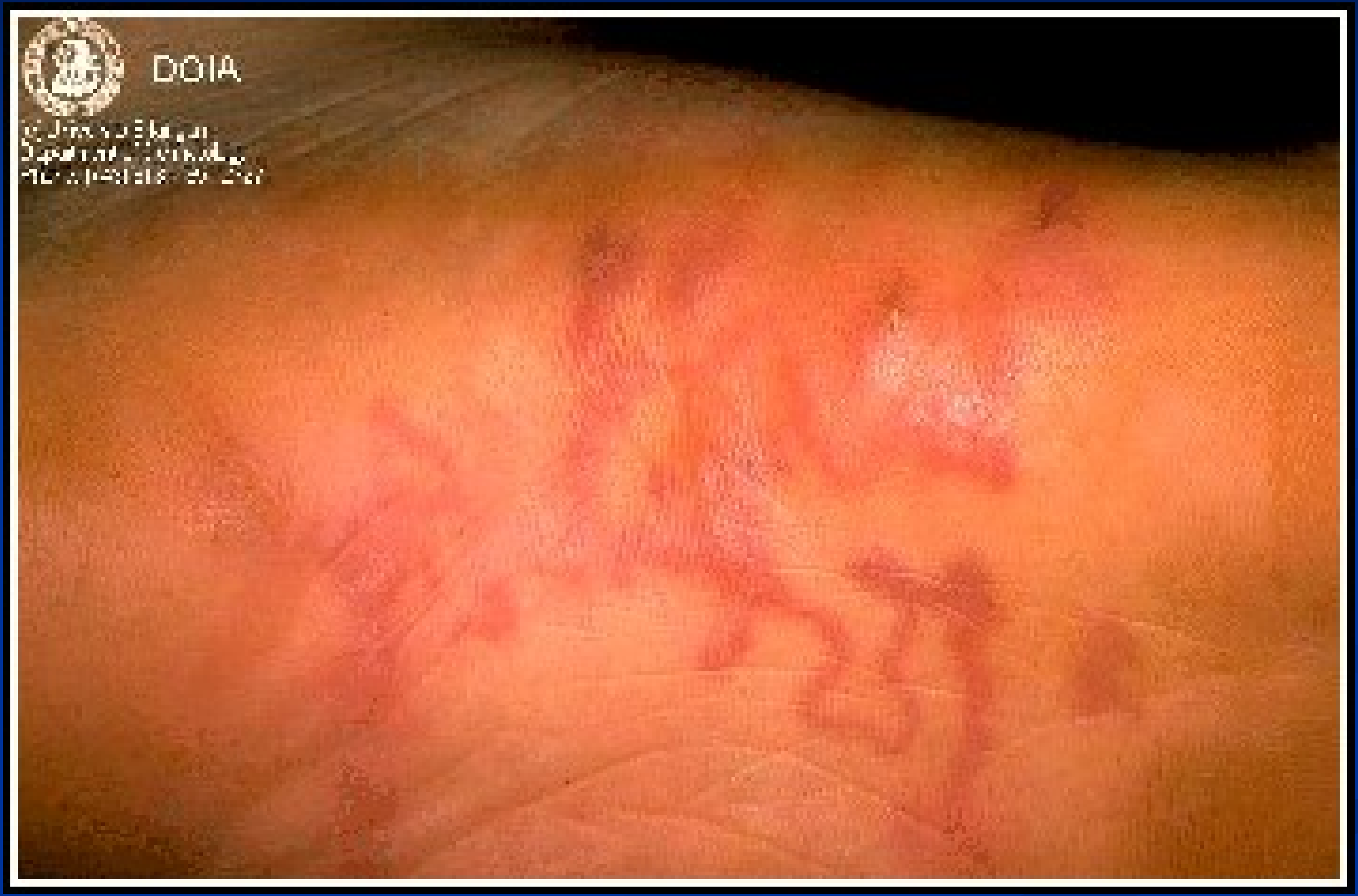
- Causative agent usually dog & cat hookworms
  - *Ancylostoma braziliense, A. caninum*
- Worldwide
- Third stage larva invades skin, remains as L3 and migrates through cutaneous tissue
- Humans are dead end host for parasite


Circulation
Larvae migrate to various organs where their development is arrested
Larvae released in intestine
In pregnant and lactating dogs, the larvae can be reactivated and cause:
- intestinal infection in the mother
- infection of the offspring (by transplacental and transmammary transmission)
Dogs > 5weeks (non-pregnant)
Human (and other paratenic hosts)
Larvae released in intestine
Circulation –> Lungs –> Bronchial tree –> Esophagus
In heavy infections, larvae can be passed in feces
Adults in lumen of intestine
♀
♂
Dogs < 5 weeks
Eggs Ingested
Eggs passed in feces
External Environment
Embryonated egg with larva
Eggs
= Infective Stage

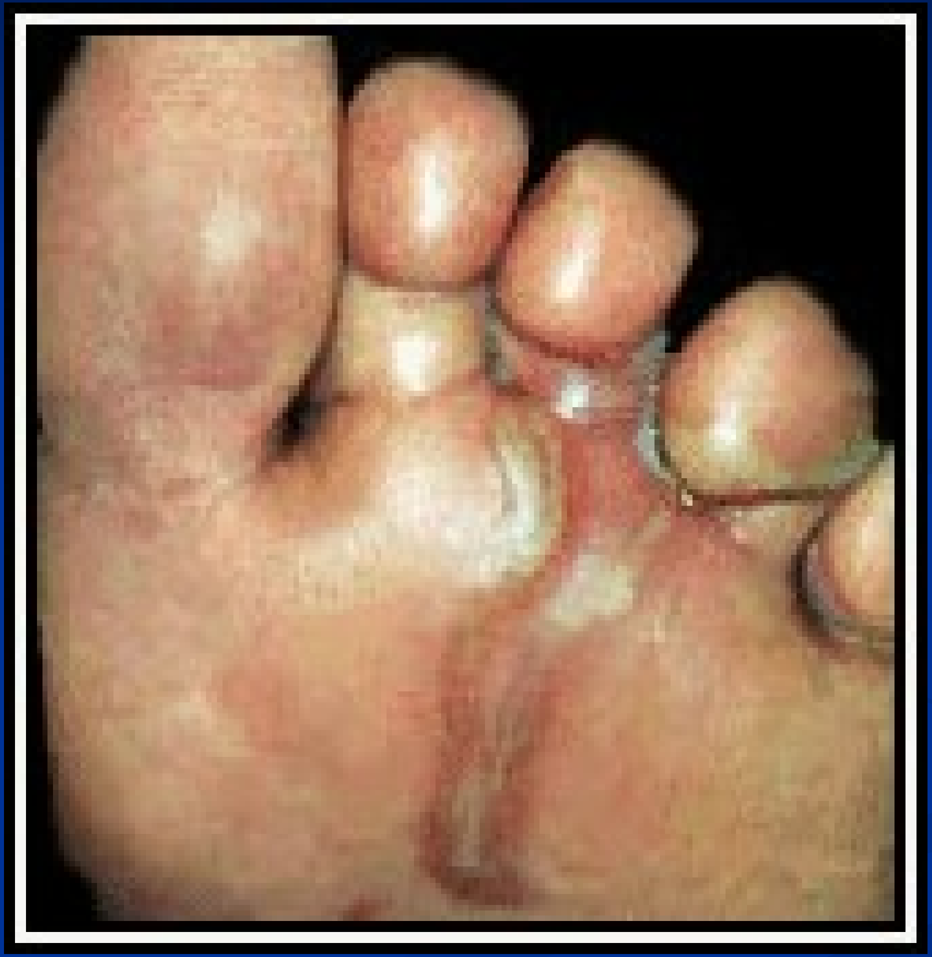
CLM (cont)

- Incubation period 1-3 days
- Larva advances several mm/day
- Can last up to a year
- Monocytic & eosinophilic reaction w/ eventual formation of eosinophilic granulomas
- Erythematous & serpiginous tracts
- Intense pruritis
- Can see vesicles, papules, desquamation

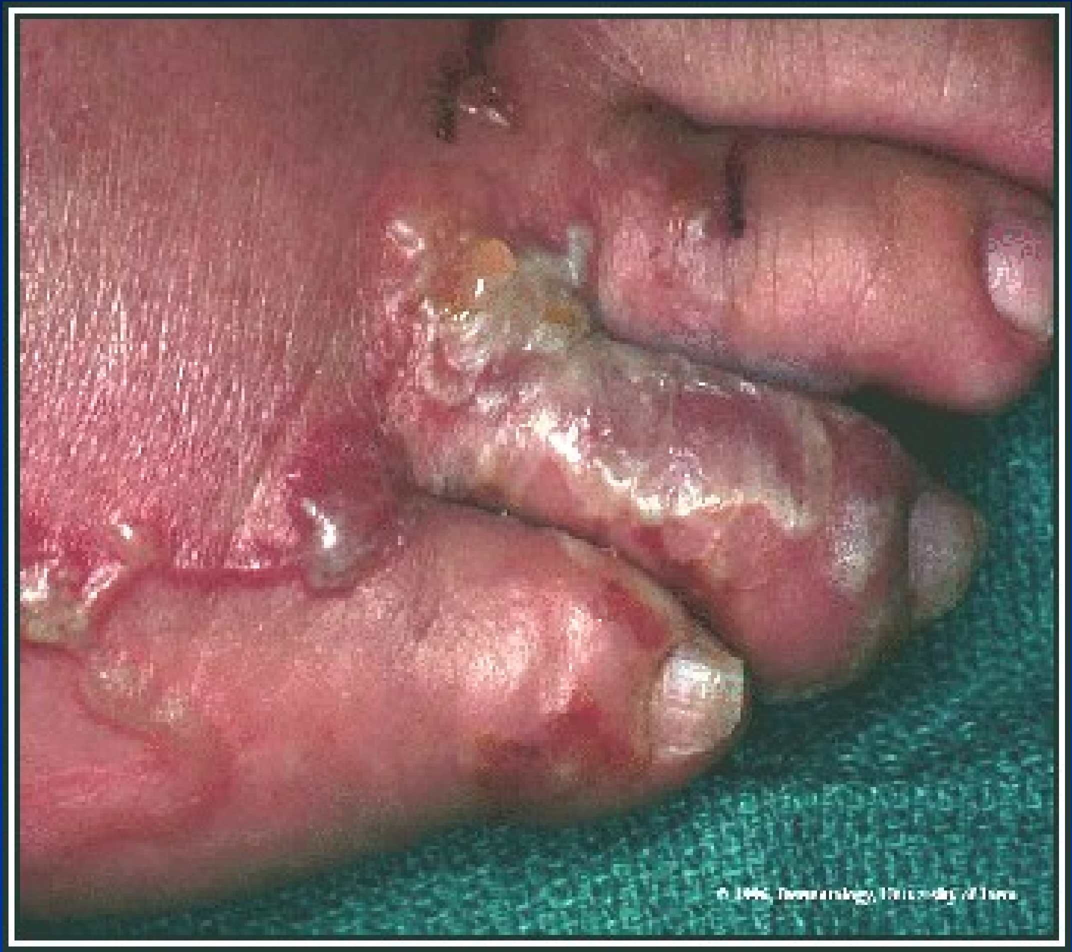
CLM, foot



CLM, foot



CLM, foot



© 1996, Dermatology, University of Iowa

CLM (cont)

- Differential
  - Impetigo, pyoderma, dermatophytoses, scabies, urticarial rxns, myiasis, ground itch from human hookworms
- Treatment
  - Ivermectin 150 µm/kg po single dose
  - Albendazole 200 mg po bid x 3d

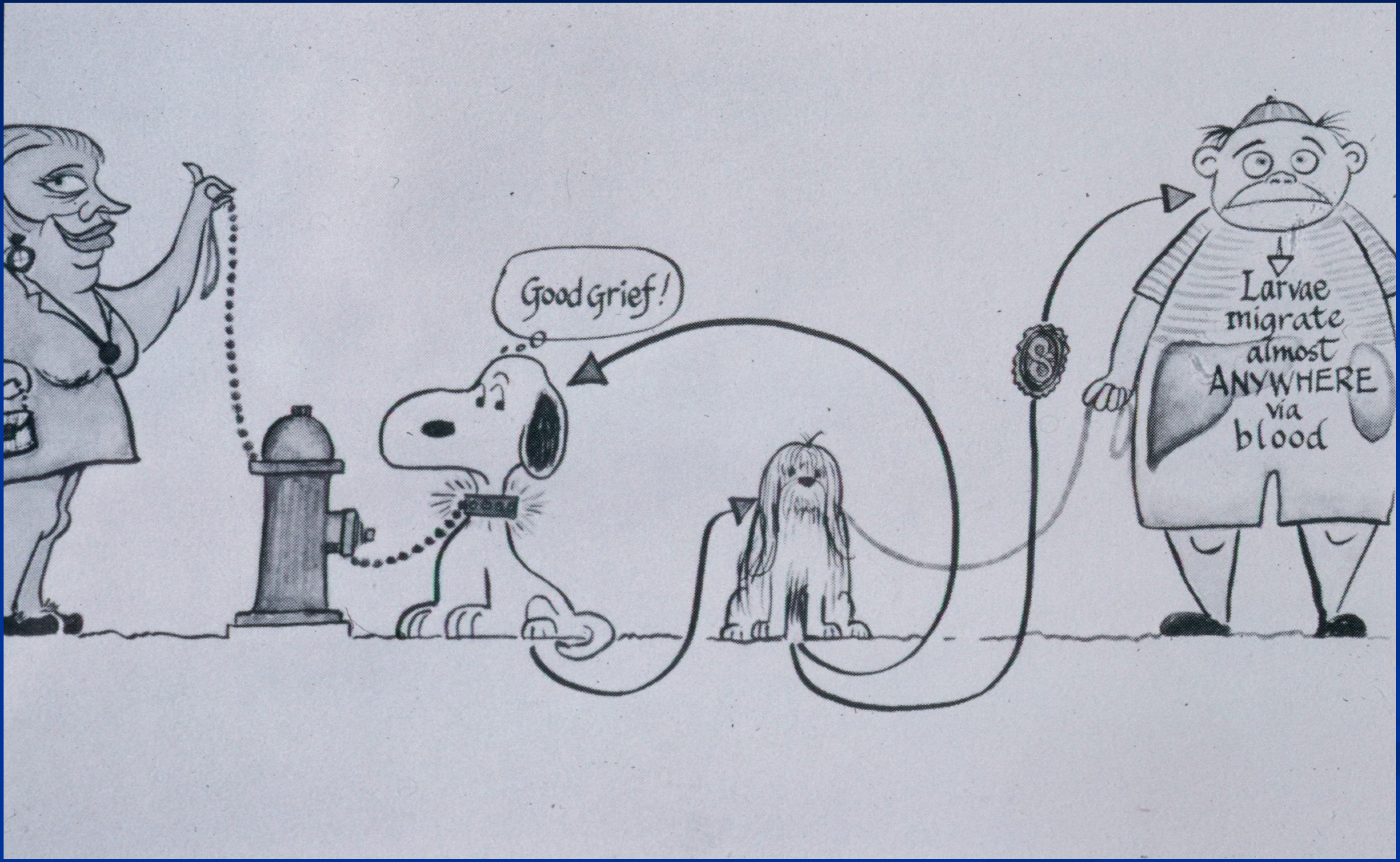
Visceral Larval Migrans Toxocariasis

- Most human cases from *Toxocara canis* (dog hookworm), few from *T. cati* (cat hookworm) & *Baylisascaris procyonis* (raccoon)
- Worldwide
- In US 2.8% prevalence of seropositivity, esp in kids (in some areas, up to 28%)
- Usually acquired via ingestion of embryonated eggs in soil contaminated with dog or cat feces
- Can be acquired by ingestion of infective larvae in meat of transport hosts (rodents, birds)

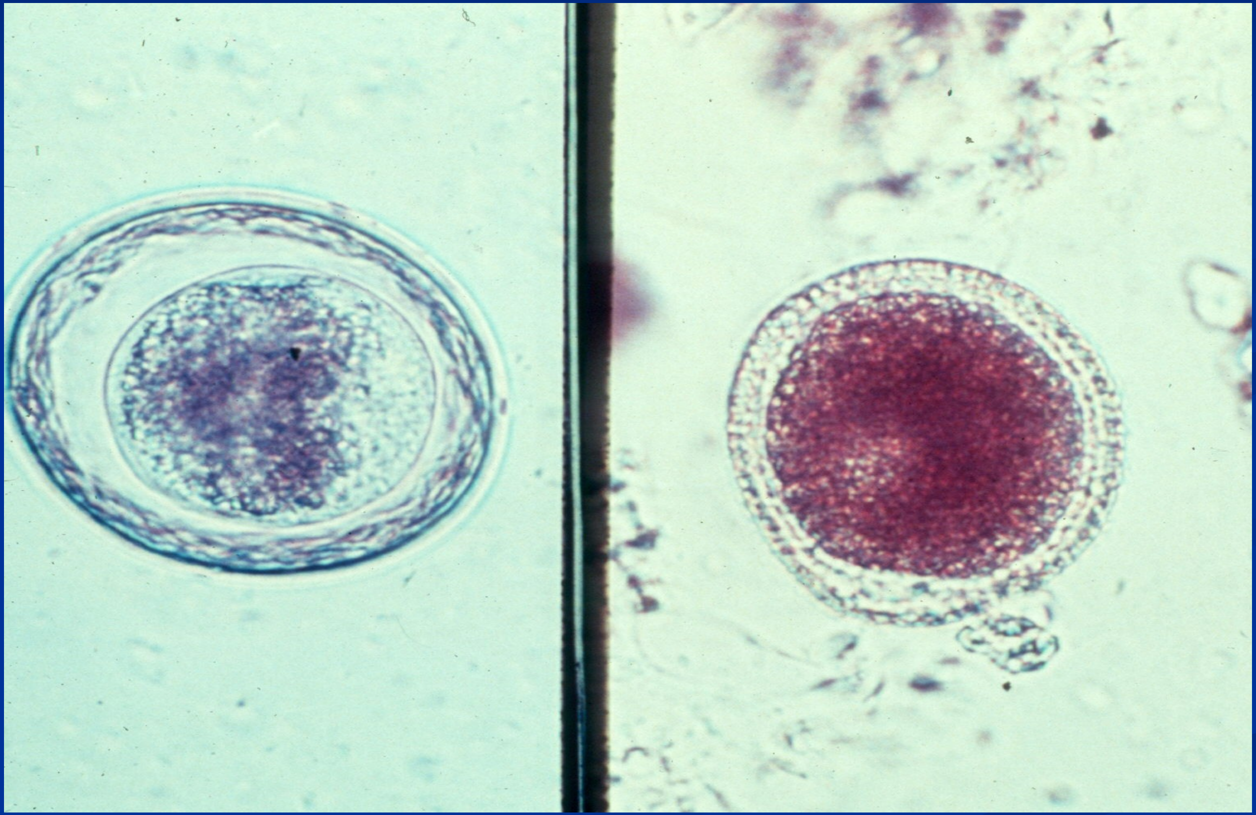
VLM Life Cycle

- Eggs in soil ingested by human
- Second stage larvae hatch
- Migrate to all tissues, esp. liver & lungs but rarely mature
- Dogs infected by ingesting eggs, eating paratenic rodents w/ larvae in tissue, transplacentally, via lactation, or mother eating vomitus & feces of infected puppies

Life cycle of *T. canis*


Good Grief!
Larvae migrate almost ANYWHERE via blood

Infective eggs found in soil



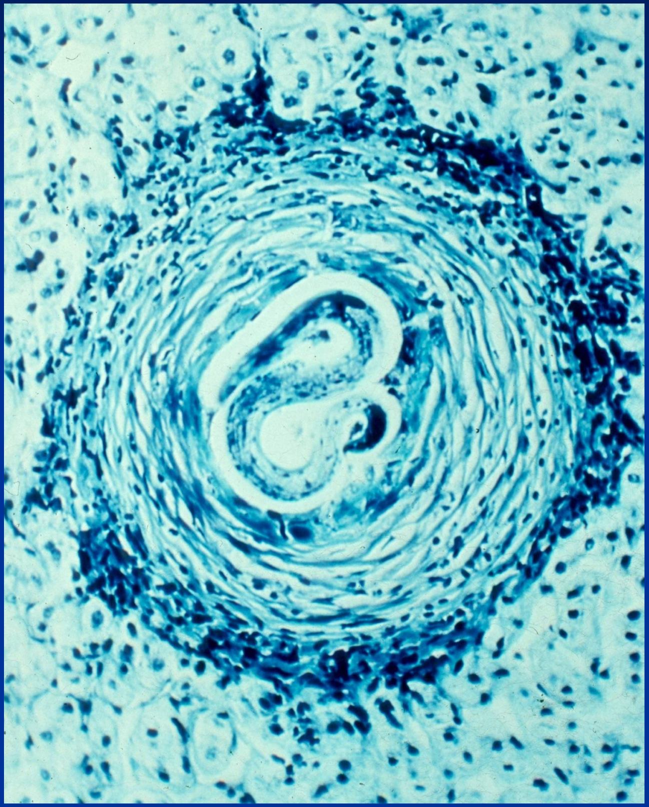
T. canis

T. cati

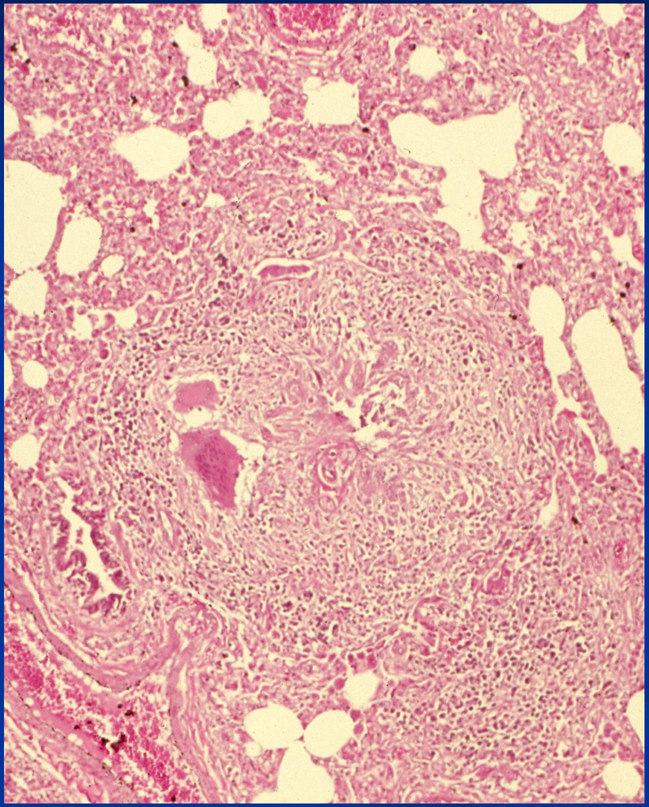
VLM Disease

- Dependent on site – liver, lungs, eye, kidneys, heart, spleen, CNS
- Mechanical damage + inflammation produce a trail of granulomas following larval migration
- Cough, wheezing, fever, anorexia, malaise, irritability, muscle pain, arthritis, pallor, eosinophilia up to 90%
- In eye, can cause retinal detachment, blindness, endophthalmitis w/ granuloma formation

Larvae in tissue



T. canis L2 in liver



T. canis L2 in lung

VLM Diagnosis

- Patient w/ persistent eosinophilia
- Biopsy
  - Eosinophilic granulomas w/wo larvae
- Serology
  - ELISA (available from CDC)

VLM Treatment

- DEC 2mg/kg po tid x 10d
- Albendazole 400 mg po bid x 5d
- Mebendazole 100-200 mg po bid x 5d
- Ocular LM
  - Within first 4 weeks, oral prednisone 30-60 mg po qd x 2wks + triamcinolone acetonide 40 mg subtenon weekly x 2wks

Baylisascaris procyonis

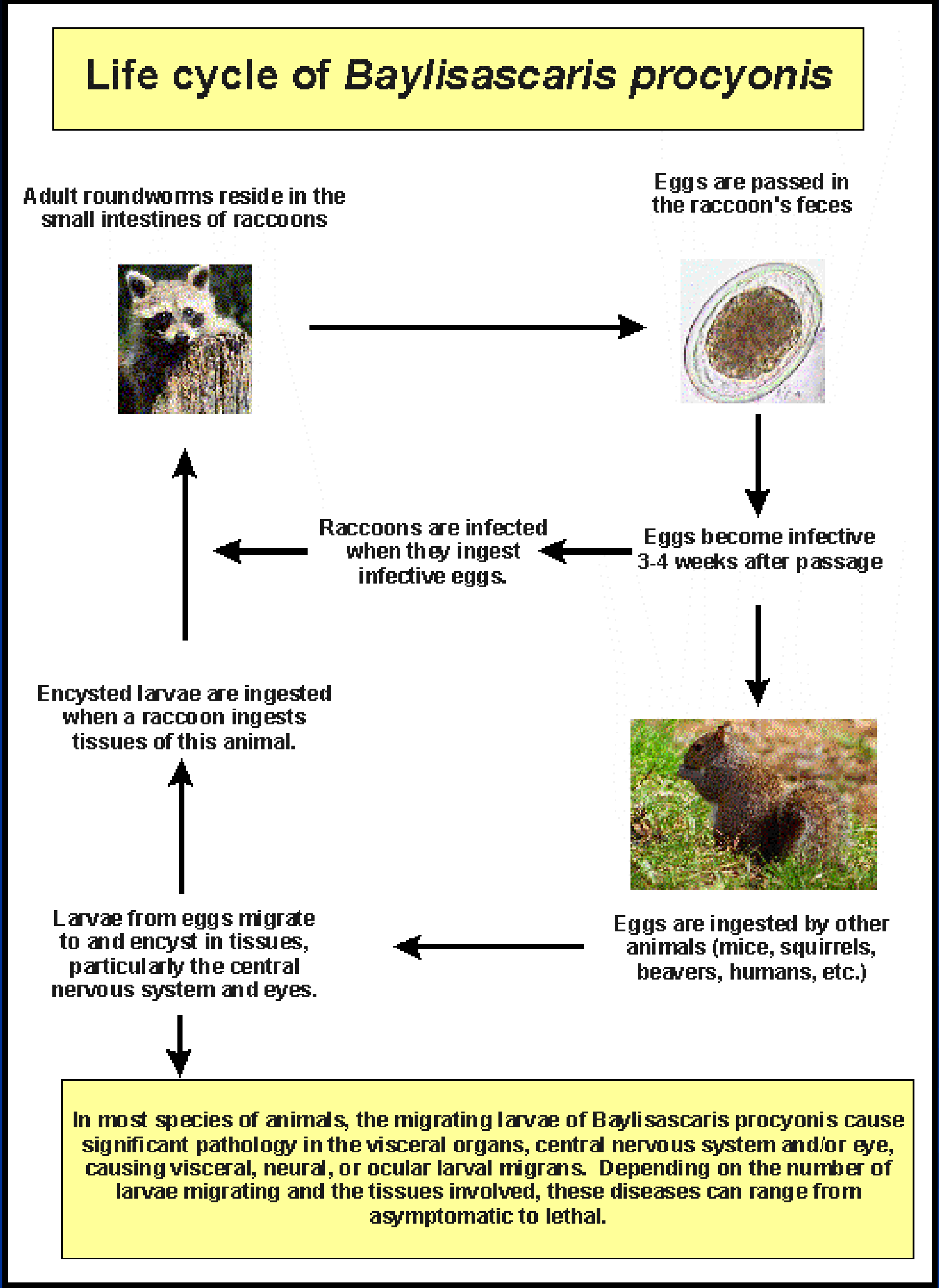
- Emerging disease from increasing exposure of humans to raccoons

Baylisascariasis

- Among the most severe of all helminthic zoonoses
- Human disease is difficult to treat & has very poor prognosis
- Usually affects children
- Raccoon is natural host (70% infection rate in adults, 90% in juveniles)

B. procyonis Biology

- Similar biology to *Toxocara canis*
- Eggs shed in feces, embryonate & become infective in 2-4 weeks
- Infected raccoons shed large amounts of eggs (20k eggs/gm feces average)
- Infective eggs swallowed by humans through pica or food contaminated w/ soil containing eggs, produces VLM syndrome
- Infectious dose approx. 5000 eggs



Life cycle of *Baylisascaris procyonis*

Adult roundworms reside in the small intestines of raccoons

Eggs are passed in the raccoon's feces

Eggs become infective 3-4 weeks after passage

Raccoons are infected when they ingest infective eggs.

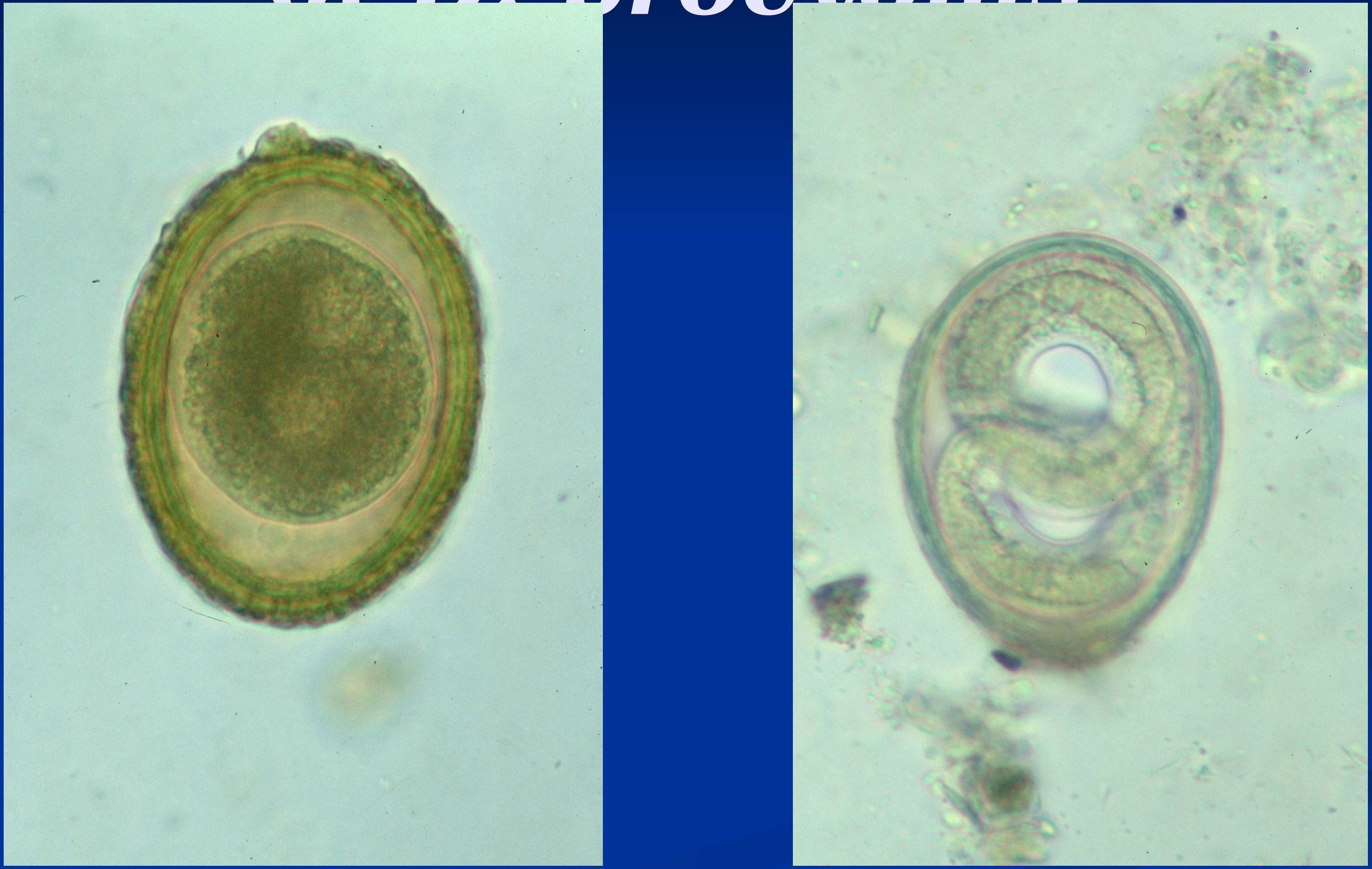
Eggs are ingested by other animals (mice, squirrels, beavers, humans, etc.)

Larvae from eggs migrate to and encyst in tissues, particularly the central nervous system and eyes.

Encysted larvae are ingested when a raccoon ingests tissues of this animal.

In most species of animals, the migrating larvae of Baylisascaris procyonis cause significant pathology in the visceral organs, central nervous system and/or eye, causing visceral, neural, or ocular larval migrans. Depending on the number of larvae migrating and the tissues involved, these diseases can range from asymptomatic to lethal.

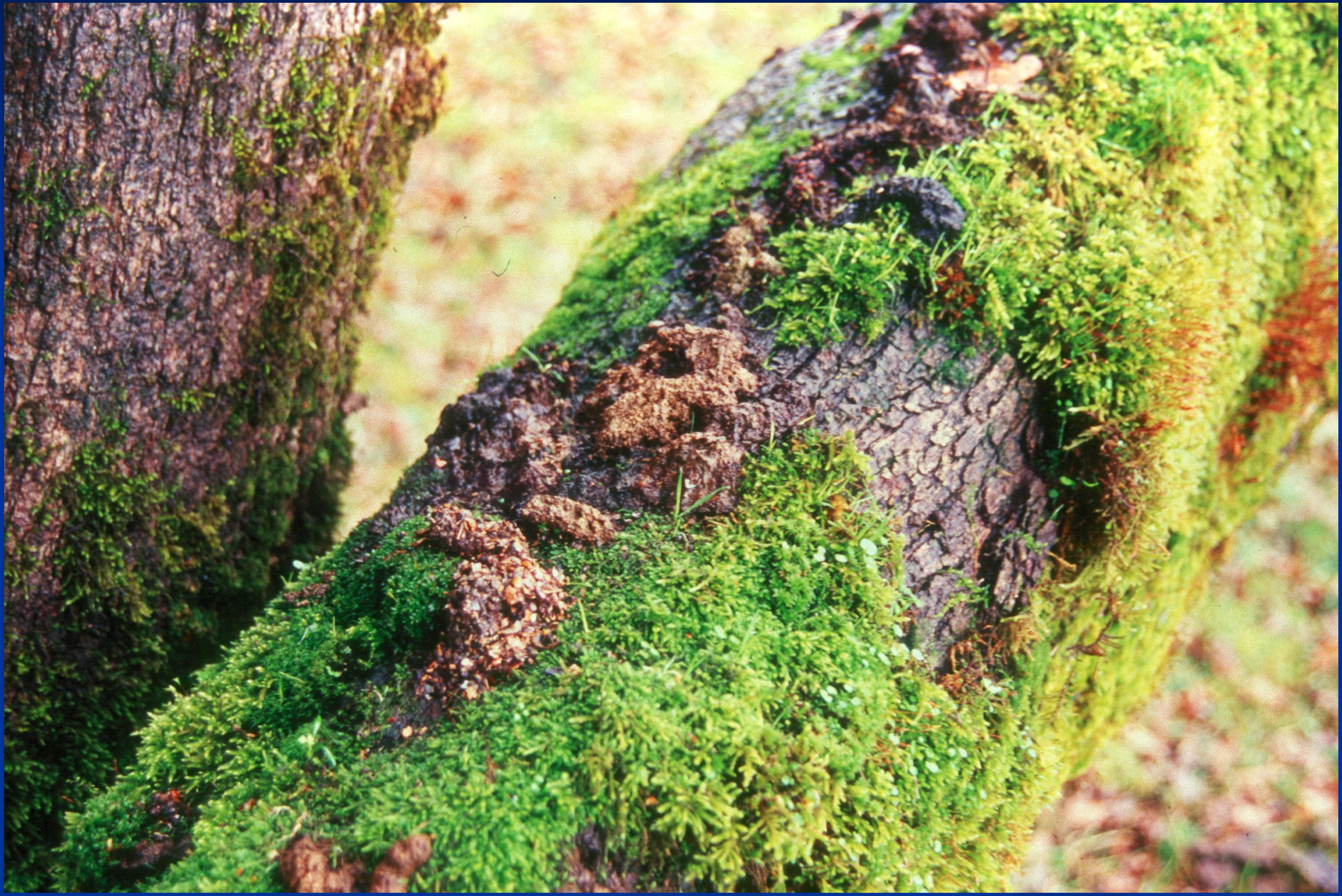
Unembryonated & embryonated infective eggs of *B. procyonis*



B. procyonis **Epidemiology**

- Raccoon ‘latrines’ in urban & periurban areas seed soil with infective eggs
- Latrines usually on logs in wild, around homes on roofs, lawns, woodpiles, fence tops, tree bases, outbuildings
- Eggs very hardy and resistant to drying, remain viable for years
- Abundance of food & shelter have greatly increased raccoon populations in urban & suburban areas

Raccoon latrine



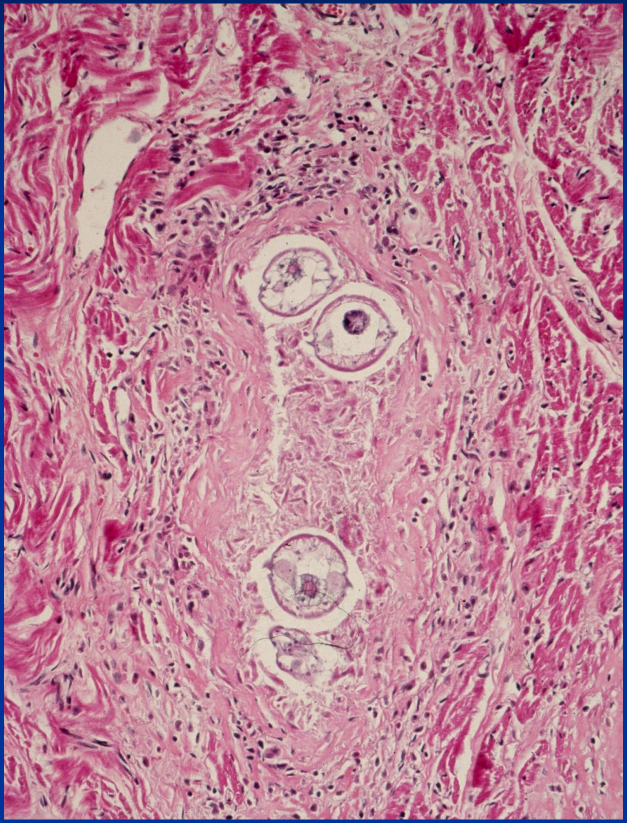
B. procyonis Epidemiology (cont)

- First case reported in 1980 in 10 mo old infant, fatal eosinophilic meningoencephalitis
- 12 cases of severe or fatal encephalitis reported in literature as of 2002, underreporting due to lack of recognition
- Dogs reported to develop patent infection, could become important source of human infection

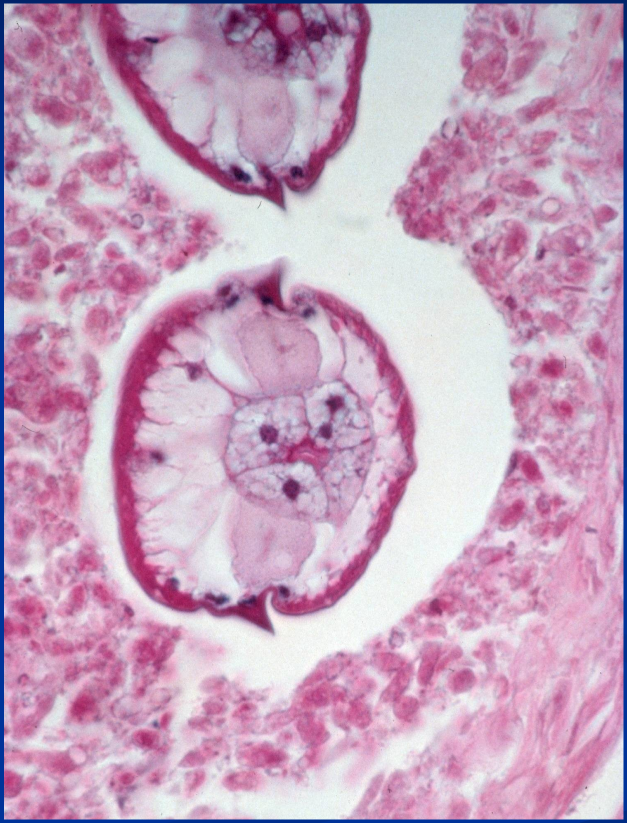
B. procyonis Disease

- Larvae have very aggressive migration behavior in all tissues but have predilection for CNS & eye
- Direct damage from migration & intense eosinophilic reaction
- IP 2-4 wks
- Ocular larval migrans (OLM)
  - Chorioretinal lesions
  - Larval tracts
  - Migrating larvae
  - Can cause diffuse unilateral subacute neuroretinitis (DUSN)

B. procyonis larvae in tissue



Larvae in cross section, heart



Larvae in lymph node, 180x

B. procyonis Disease (cont)

- Disease severity related to
    - number of eggs ingested
    - extent of larval migration
    - severity of inflammation
    - amount of tissue necrosis
    - length of delay before tx instituted

Baylisascariasis Diagnosis

- O&P of no use, as worms don’t mature to produce eggs
- Eosinophilic pleocytosis in CSF + peripheral blood eosinophilia in patients w/ progressive neurologic deficits characteristic but not pathognomonic
- Differentiate from toxocariasis w/ negative serology
- Biopsy is hit or miss
- Serology for *Baylisascaris* not routine but available from Purdue Univ Dept of Vet Pathobiology

Baylisascariasis Treatment

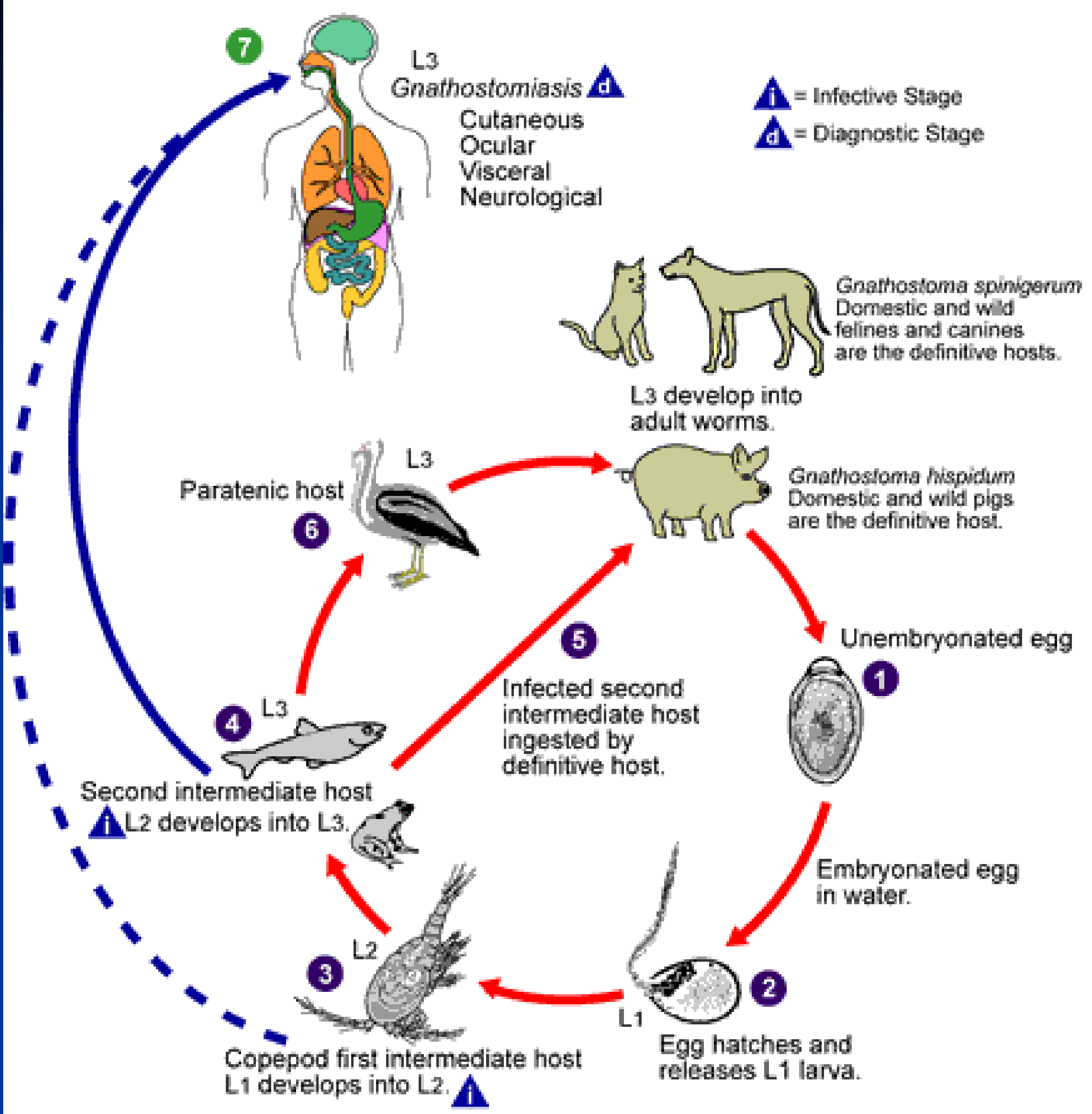
- No treatment in literature has changed prognosis
- Poor prognosis usually due to late start of tx as larvae continue aggressive migration
- Albendazole drug of choice, best to start tx on presumptive diagnosis, rather than wait for definitive results
- DEC shown to be effective in experimentally infected mice

Gnathostomiasis

- *Gnathostoma spinigerum*
- Southeast Asia, Israel, Central and South America
- Acquired from ingesting raw or poorly cooked fish, fowl, amphibians, & rarely pork
- Normal definitive hosts are dogs, cats, other fish-eating mammals
- Humans dead end host

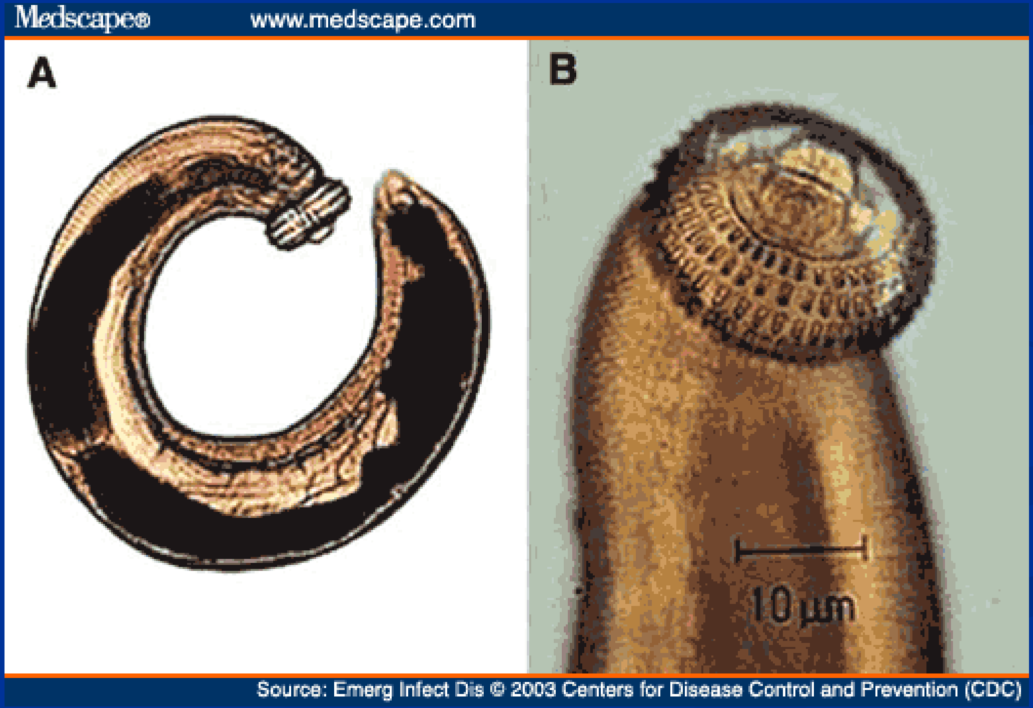
Gnathostomiasis Life Cycle

- Eggs shed via feces into water & hatch
- Ingested by copepod, molt to second stage
- Infected copepod ingested by 2nd intermediate host (frogs, fish, reptiles, birds), larvae molt to third stage & encapsulate
- Eaten by mammal, larvae penetrate stomach, pass through liver, wander about, get back to stomach, form tumor-like mass, mate & produce eggs


⑦
L3
Gnathostomiasis d
Cutaneous
Ocular
Visceral
Neurological
i = Infective Stage
d = Diagnostic Stage
Gnathostoma spinigerum
Domestic and wild felines and canines are the definitive hosts.
L3 develop into adult worms.
L3
Paratenic host
6
Gnathostoma hispidum
Domestic and wild pigs are the definitive host.
5
Infected second intermediate host ingested by definitive host.
Unembryonated egg
1
4
L3
Second intermediate host
i L2 develops into L3.
Embryonated egg in water.
L2
3
L1
2
Egg hatches and releases L1 larva.
Copepod first intermediate host
L1 develops into L2. i

Gnathostomiasis Disease

- Same as other LM syndromes
- Edema, hemorrhage, eosinophilic & plasma cell infiltrates, necrosis, granuloma formation
- Epigastric pain, nausea, vomiting, anorexia, pruritis, urticaria, fever, eosinophilia up to 90%
- CLM form – larvae migrates 1cm/hour



Source: Emerg Infect Dis © 2003 Centers for Disease Control and Prevention (CDC)

Gnathostomiasis Treatment

- Surgical removal

- Albendazole 400 mg po qd or bid x 21d